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EDWARD L. JENKINSON

Caldwell Lecturer, 1954

THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 73 JUNE, 1955 No. 6

THE PYLORIC ANTRUM OF THE STOMACH

CALDWELL LECTURE, 1954*

By EDWARD L. JENKINSON, M.D.

Professor and Chairman of the Department of Radiology, Northwestern University Medical School; Director of the Department of Radiology, St. Luke's Hospital

CHICAGO, ILLINOIS

Mr. President, members of the American Roentgen Ray Society and guests:

I WISH to thank Dr. Kirklin for his very generous introduction and his kind remarks, and to thank President Dickinson for inviting me to deliver the Caldwell Lecture. It is a great honor and I am not unmindful of the responsibility it entails.

I am cognizant of the great men who have preceded me as Caldwell Lecturers and when I look over this list I realize my inadequacy and I only hope that in a small way I may measure up to their stature.

It is a privilege to again join with members of the Society and friends of Dr. Caldwell to pay tribute to one of the great martyrs of Medicine. We are gathered here this evening primarily to pay our respects to the memory of Dr. Eugene Caldwell.

Dr. Caldwell sacrificed his life in the advancement of science, especially in the development of Roentgenology. He was a rare combination endowed with great mechanical and electrical knowledge, as well as with an investigative interest in the medical application of the roentgen rays.

His early training as an engineer provided knowledge of the physical equipment and his medical training stimulated his inventive mind in the application of the roentgen rays in the study of disease.

I knew Dr. Caldwell only slightly but was well aware of his many early contributions in engineering and medicine, especially roentgenology. We are paying tribute to his memory by carrying on his work in roentgenology, which he loved so much.

I think it might be appropriate at this time to give a brief history of the Caldwell Lecture. The idea of the Lecture originated in 1920 with Dr. James T. Case who was then President of the Society. Dr. Case felt that he would like to experiment with a lecture allowing the speaker ample time to fully cover his subject. On numerous occasions when listening to teachers attempting to cover very complex problems in fifteen or twenty minutes, for lack of sufficient time they were required to hurry and incompletely cover their subject. His

* Delivered at the Fifty-fifth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., Sept. 21–24, 1954.

idea was to allow one speaker, each year, ample time to present his paper. In consultation with other Officers of the Society and particularly with Dr. Preston M. Hickey, an affirmative decision was made, and the Lecture was named in honor of Dr. Eugene Caldwell.

Dr. Caldwell died on June 20, 1918, after many surgical operations for malignant changes due to his early experimentations with the roentgen rays. For anyone interested in the life of Dr. Caldwell, I recommend The Caldwell Lecture, 1928, by Preston M. Hickey published in the February, 1931, number of the AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY.[52]

The first Caldwell Lecture was delivered by Dr. Walter C. Alvarez at Minneapolis, Minnesota in September, 1920. Dr. Alvarez discussed "Peristalsis in Health and Disease." The lecture was very interesting and educational, and was the forerunner of much of his exhaustive work on the digestive tract.

Our subject for tonight "The Pyloric Antrum of the Stomach" offers a wide and much discussed field for study. I did not realize how much has been written about the pyloric antrum until I began a review of the recent literature. Although many articles have been written by many authors there is no unanimity of opinions as to the value of the clinical, gastroscopic or roentgenographic findings in arriving at a positive differential diagnosis. Most authors summarize their papers by stating that even though all available examinations have been carefully carried out by competent men it is often necessary to resort to surgery before a diagnosis can be made. Opening the abdomen and palpating the pyloric antrum is not enough. The stomach should be opened, inspected and palpated and frozen sections made. If it is not possible to make frozen sections and the lesion is still questionable, the antrum should be resected.

I will discuss in some detail the embryology, anatomy and the common lesions of the pyloric antrum such as hypertrophy of pyloric muscles, antral gastritis, prolapse of the mucosa, deformities and narrowing of the pyloric antrum due to adhesions and bands, "extragastric" and "intragastric" lesions, carcinoma of the pyloric antrum and ulcer of the pyloric antrum.

The dilating portion of the foregut which develops into the stomach is at first attached ventrally and dorsally by a mesentery or membrane. The posterior border of the primitive stomach begins to dilate, and grows very rapidly. This portion of the stomach dilates and rotates to the left and downward, so that the lesser curvature of the stomach represents the comparatively unchanged ventral wall of the original foregut whereas the greater curvature is the dilated portion. The stomach then from being sagitally suspended becomes in rotation placed so that the left surface is anterior and the right posterior, and the ventral attachment of the mesentery merges into the gastrohepatic mesentery. The cul-de-sac is formed by the dorsal mesentery which is located behind the stomach and the mesentery sags to become the greater omentum. There is a fusion of the dorsal mesentery of the duodenum and the pyloric end of the stomach with the posterior wall of the abdomen and so attaches the duodenum firmly and carries the pancreas to a retroperitoneal position. From the 10 to 20 millimeter stage, that is forty-two to fifty-five days of the embryo, the pyloric end of the stomach is relatively long, being about one-half the entire length of the stomach. Later it dilates and becomes relatively shorter. Two folds of the mucous membrane from the cardiac end of the stomach to the pylorus along the lesser curvature are distinctly discernible about this time. They form the gastric canal.

The stomach is shaped somewhat like a cone, the base of which contains its cardiac orifice. The right margin of this orifice continues with the lesser curvature of the stomach and extends from the cardiac to the pyloric orifice. Near the pyloric end is a

well marked notch called the incisura angularis, which varies with the position and size of the stomach, but which serves as an anatomical landmark to divide the stomach into the right and left portions. The gastrohepatic ligament is attached along the lesser curvature and contains the left gastric artery and the right gastric branch of the hepatic artery. The greater curvature is between four and five times longer than the lesser curvature. On the greater curvature, opposite the incisura angularis, there is a dilatation and to the right of this a slight groove. The part of the stomach between this groove, called the sulcus intermedius, and the duodenum is the pyloric antrum. Outlines of the stomach vary greatly, chiefly due to the quantity of its contents, but also due to some extent to the position of the body and to the type of the individual. Usually a short, heavy man has a stomach located in the transverse diameter, commonly called the steer horn type, whereas the long enteroptotic type of individual has the long fishhook type of stomach.

For practical purposes, a satisfactory outline is that suggested by Cannon who divided the stomach into two main portions, the cardia and the pylorus. This division is indicated on the lesser curvature by the incisura angularis. The portion of the stomach to the left of the line from the incisura to the greater curvature is called the cardiac portion, and that to the right of this line is the pyloric portion. The cardiac or the left portion is again subdivided into the body of the stomach, or the pars media, as it is often called, and the fundus. The fundus lies above a line drawn horizontally through the cardiac orifice, and between the fundus and the incisura angularis is the body or the pars media.

To the right of the incisura angularis is the pyloric portion of the stomach which is subdivided by some observers into three parts, and by others into two. Next to the pyloric sphincter is the pyloric canal, about $1\frac{1}{4}$ inches (3 cm.) long. The remaining portion of the pyloric division, from the pyloric canal to the incisura, is the antrum of the stomach, sometimes divided into the antrum and the vestibule, the latter division beginning at the incisura while the antrum is between the vestibule and the pyloric canal. However, this subdivision seems unnecessarily complicated and the antrum may be considered as all of the stomach between the incisura angularis and the pyloric canal.

Except along the upper and lower borders, the stomach is covered with peritoneum and between this is a muscular tunic divisible into three laminae of smooth muscle. The outer or longitudinal layer of muscle fibers is a continuation of the external longitudinal muscular lamina of the esophagus. It forms a narrow and powerful band along the lesser curvature with a broader and feebler band along the greater curvature. Along the lesser curvature of this bundle of muscle spreads out somewhat in the pyloric region and becomes thinner. The superficial layer of the longitudinal muscle fibers is practically lost in the pyloric portion of the stomach, except along the lesser curvature where a few of the fibers pass into the duodenum. A second set of longitudinal fibers arises on the body of the stomach, passes over the pylorus and deeper fibers, to become interlaced with the circular fibers of the pyloric sphincter. The second of the muscular layers of the stomach is circular and is arranged in bands covering all of the stomach except the fundus. This is a continuation of the circular laminae of the esophagus and duodenum. At the pylorus this layer is quite powerful and forms an aggregation of circular muscle fibers which constitute the pyloric sphincter. The transition between the circular fibers of the stomach and the duodenum is sudden. The circular fibers of the duodenum are much weaker than those of the pylorus. The third, or deepest layer of muscles of the stomach is the oblique lamina. At the cardia this layer is connected with the circular muscle fibers of the esophagus. The oblique layer diverges upon the anterior and pos-

terior gastric walls but along the lesser curvature the fibers run almost parallel and go to the pyloric portion of the stomach. At the greater curvature, the fibers take the direction of the circular muscle fibers, which they reinforce, but around the stomach they pursue an oblique course. The gastric mucosa is separated from the muscular layer by the tela submucosa. The mucous membrane is brownish-red and more pinkish in the pyloric than in the cardiac regions. The color contrasts very sharply with the light color of the mucous membrane of the esophagus. The mucosa and the submucosa form numerous folds of rugae when the stomach is empty which are smoothed out when the stomach is distended. They are disposed as an irregular network except along the lesser curvature where the mucosa is arranged so as to bound a longitudinal groove. However distended the stomach may be, there is a circular fold of the mucosa at the pylorus which is forced forward by the contraction of the pyloric antrum and canal. There are also numerous elevations in the gastric mucous membrane which are incompletely separated from each other. Over the entire gastric mucosa small depressions can be seen with a hand lens. They have a honey-combed appearance and constitute the ducts of the gastric glands. At the bottom of these depressions there are one or more orifices, the openings of the tubules from the glands. Between these depressions there are small, flat, villous processes, more marked in the pyloric region.

The three muscular layers of the stomach are united by thin partitions of connective tissue. In the connective tissue between the longitudinal and circular muscle layers are the myenteric nerve plexus or Auerbach's plexus and the larger blood vessels. The submucosa is a loose areolar tissue supporting blood vessels, lymphatics and submucous plexus of nerves (Meissner's plexus), which are distributed to the mucous membrane. The arterial blood supply of the stomach consists of the left gastric artery from the aorta, the right gastric and right gastro-epiploic branches from the hepatic artery, and the left gastro-epiploic and the short gastric arteries of the splenic artery. After supplying various other structures of the stomach, these branches anastomose freely in the submucosa, forming an extensive arterial plexus and are distributed to the mucous membrane. From the submucous plexus the branches penetrate the mucosa making a dense capillary plexus enclosing the secreting gastric glands. The veins pass between the glands to the submucosa, emptying into the splenic veins and into the coronary vein that runs along the lesser curvature of the stomach, thence into the superior mesenteric vein or directly into the portal vein. A plexus is formed in the submucosa by the veins.

Lymphatics begin as loops or as delicate dilated tributaries between the gastric glands and form an extensive plexus near the basal border of the mucous membrane. From this plexus lymphatic channels penetrate the lamina muscularis mucosae making a broad submucous plexus from which lymphatics penetrate the muscular layers, finally reaching the lymph nodes along the greater and lesser curvature.

The nerve supply of the stomach is derived from the vagi and from the sympathetic system through the splanchnic nerves, entering along with the blood vessels. These two types of nerves form plexuses, the myenteric or Auerbach's plexus, between the longitudinal and circular muscular layers, and the submucous or Meissner's plexus in the deeper part of the tela submucosa. Apparently both of these plexuses contain ganglionic elements and small ganglia may be found at the intersections of their branches. The submucous or Meissner's plexus is made up of smaller fibrils than Auerbach's plexus. From the submucous plexus the fibrils pass through the mucosa and terminate in and around the walls of the blood and lymphatic vessels. Minute branches also go to the epithelium of the secreting glands, ending upon the cells as varicose fibrils. The mucosa supports the mucous membrane. The

deepest layer of this mucosa is in the muscularis mucosa, forming a thin but complete layer from one end of the stomach to the other. The lamina muscularis mucosa is divisible into an inner circular and an outer longitudinal component. Occasionally muscle fibers from the mucosa extend into the corium or supporting connecting tissue of the gastric glands. Tall columnar epithelium lines the mucosa superficially. In the corium and mucosa are closely packed tubular glands opening by wide-mouthed, crypt-like ducts.

The structure of the gastric glands varies in different portions of the stomach. The glands are usually classified as fundic, cardiac and pyloric. The fundic glands are tubular, somewhat branched and terminate in short ducts or crypts. Their secreting portion is relatively long. Several of these glands, by a short constricted portion called the neck, open into the bottom of the wide ducts or crypts which are lined with tall columnar epithelium with clear cytoplasm and nuclei at the base of the cells. The epithelium forms a lining of the mucosa of the stomach and from these cells is secreted a clear mucus-like fluid. The fundus or the secreting portion of these glands is from five to eight times as long as the duct, quite different from the fundus of the pyloric glands. The lumen of the glands is very narrow. In the fundus of the glands are two different kinds of cells, the chief cells and the parietal cells. The chief cells are relatively more abundant in the deeper portion of the fundus where there are but few parietal cells which are crowded away from the lumen toward the basement membrane. Toward the neck of the gland the parietal cells are more abundant and gradually approach nearer the lumen until in the neck of the gland the parietal cells have considerable free surface on the lumen.

The chief cells are sometimes called central or peptic cells; they are cuboidal or pyramidal in shape, with a granular cytoplasm. The nucleus is situated near the base of the cell and is spheroidal. The distal portion of the cell is the more granular part but the amount of the granular zone depends upon the degree of activity of the gland. The granules which consist of zymogen, seem to accumulate during rest and largely disappear with secretory activity. The whole cell becomes shriveled after prolonged secretion, but swells after periods of rest so that the lumen of the glands may be obliterated. Near the base of the cells filaments of prozymogen can be demonstrated. These chief cells secrete pepsinogen, and so form the pepsin of the gastric juices.

The other secreting cells of the gastric glands are the parietal cells. They are ovoid or pyramidal in shape and frequently contain two nuclei. The cytoplasm stains readily with acid dyes. The nuclei, which are spherical, are commonly situated centrally and contain much chromatin. The cytoplasm is homogeneous or finely granular. The shape of these cells varies somewhat with their location. In the fundus of the gland they are crowded out by chief cells, forced toward the basement membrane; here they are ovoid or triangular in outline with the broad base resting upon the basement membrane. Toward the middle of the tubule the parietal cells come closer to the lumen; as they are compressed by the chief cells around them they become taller. In the fundus of the tubule, where the parietal cells are remote from the lumen, and extensive system of secretory, canaliculi surrounds the cells like a net. This pericellular system carries the secretion to the lumen. The hydrochloric acid of the gastric juice is supposed to be secreted by these parietal cells, though it is believed that they actually secrete chlorides which are converted into hydrochloric acid when the secretion reaches the crypt of the ducts.

HYPERTROPHY OF THE PYLORIC MUSCLE (FIG. 1 AND 2)

In a four year survey of congenital defects in the newborn in the city of Philadelphia, 275 cases were studied. Of these, 35 had gastrointestinal tract deformities. Of this group, 20 had pyloric stenosis. In

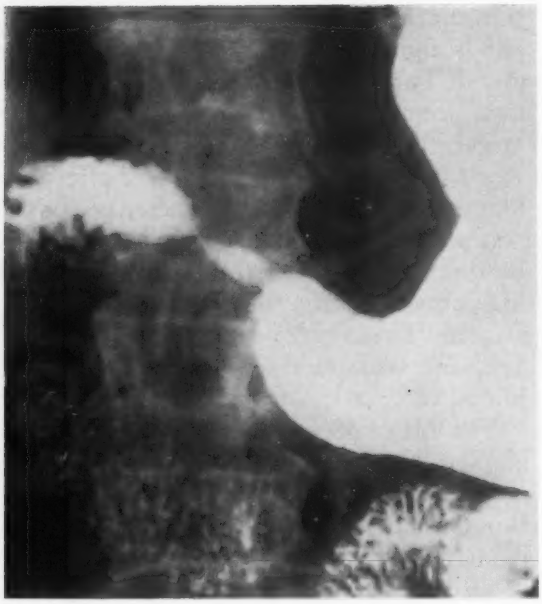

FIG. 1. Patient L. F. Hypertrophy of the pyloric muscle and redundant mucosa at operation.

the infant this condition is usually associated with no other gastrointestinal disease. The hypertrophied pyloric muscle forms a firm, almost cartilaginous mass producing variable degrees of obstruction. Usually, if untreated, the condition becomes progressively more complete and the symptoms more severe. The stomach proximal to the obstruction dilates and secretes an excess of mucus which frequently forms a plug in the pylorus to complete the obstruction. The normal infant pylorus varies in length from 1.0 to 1.5 cm.; the hypertrophied pylorus varies from 1.5 to 3.0 cm. A normal infant pylorus varies in width from 0.05 to 0.25 cm.; the hypertrophied pylorus varies from 0.3 cm. to 0.7 cm. Microscopically, the changes from normal are limited to submucosa and the circular muscle; the submucosa is usually thickened by collections of edema and fluid. The circular muscle is hypertrophied with no concomitant increase in thickness of the longitudinal muscle or the connective tissue coat. In the adult form of hypertrophy two distinct types are seen: idiopathic and concomitant. In the idiopathic type no other demonstrable pathology is found within the gastrointestinal tract; in the concomitant type, other pathology is found such as duodenal or gastric ulcer, or chronic gastritis with superficial ulceration. In either case, the circular muscle is hypertrophied just as in the infantile form with elongation and narrowing of the pyloric orifice.

It is necessary to recall the anatomy of the pylorus to understand better this localized pathologic entity. The arrangement of the circular muscles of the pylorus is such that in over 26 per cent of the subjects there is complete break between the circular muscle of the pylorus and the circular muscle of the duodenum. The longitudinal muscle, however, is more variable; about 50 per cent of the longitudinal fibers interdigitate with the circular muscles to form the dilator muscle of the pylorus, while less than 25 per cent continue into the duodenum. Thus, the pylorus is a complete sphincter of both the dilator and constrictor muscle. The bulk of the circular muscle is four or five times greater than that of the longitudinal muscle. The circular muscle has connective tissue septa which run perpendicular to the long axis of the pylorus. These divide the circular muscle into numerous closely associated units. Cross sections made through the pylorus at different levels reveal variations in the thickness of the circular muscles because of these septa. The continuity between the submucosa and the circular muscle is established via these connective tissue septa; this undoubtedly accounts for the submucosal edema in hypertrophy of the pyloric muscle. On the other hand, the connective tissue septa rarely ever penetrates the longitudinal muscle. This may explain the lack of edema in this layer in hypertrophy of the pylorus. Horton concluded from his detailed histologic studies that the complete separation of the circular muscle of the pylorus from the circular muscle of the duodenum has physiologic significance in that this separation causes a block in the antral wave as it reaches the pylorus. He felt that the chang-

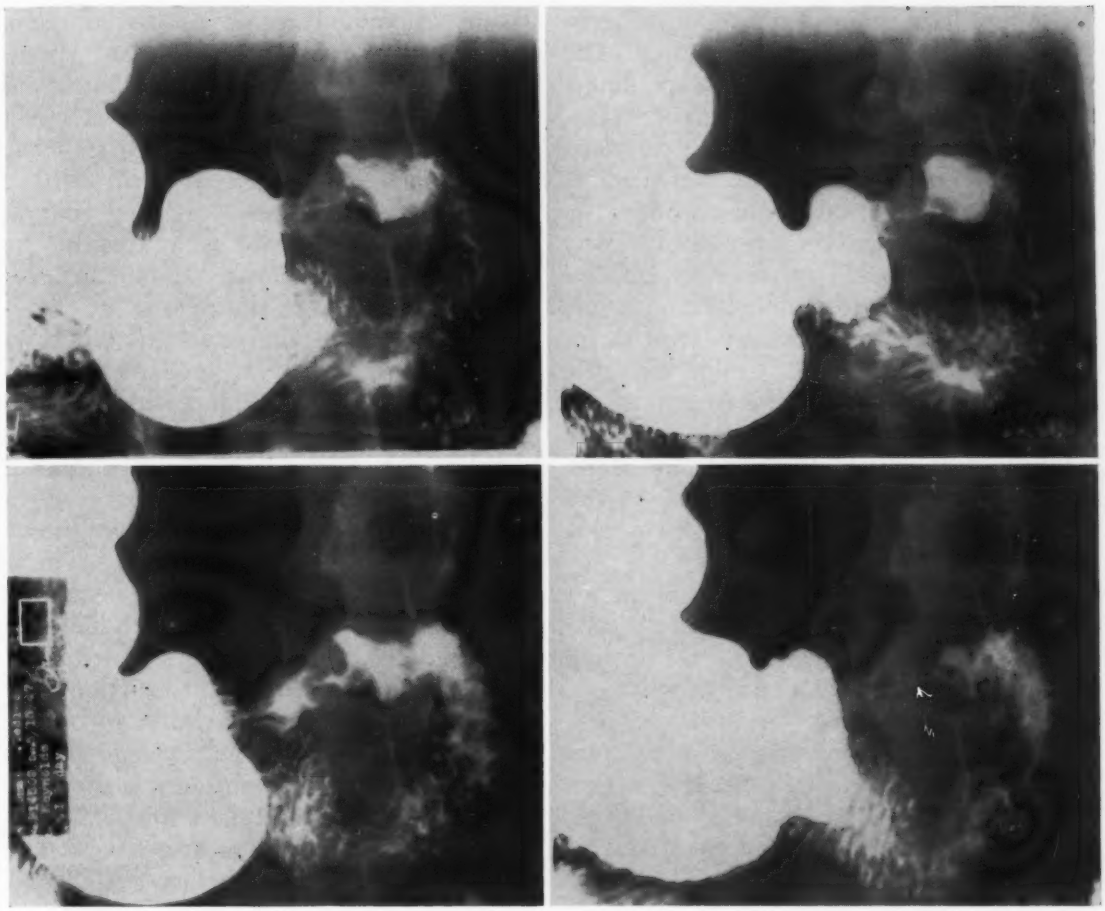

Fig. 2. Patient T. Y. Hypertrophy of the pyloric muscle, proved at operation and by sections.

es in tonus of the duodenal musculature are due to a spread of some influence along the myenteric plexus or along the narrow bridge of the longitudinal muscle connecting the duodenum and the stomach.

Fabricius Hildanus is given credit for first describing and recognizing hypertrophy of the pyloric muscle. Wolfgang Helfer, in 1675, reported that the condition was very rare and that he himself had never seen a case. Early reports were made by Blair in 1717, Weber 1758 and Armstrong in 1771. Hezekiah Beardsley, in 1788, reported on "congenital hypertrophic pyloric stenosis" found at autopsy in a boy five years of age. Jean Cruveilhier, in 1835, reported a case in a female seventy-two years of age who had vomited all her life; at autopsy she was found to have a hypertrophy of the pyloric muscle. A typical case was reported by Williams in 1841. Siemon-Daworsky reported 10 adult cases in 1842. The term "congenital pyloric stenosis" was applied to the condition in 1879 by Landerer. Thirty-one cases were reported in 1885 by Maier studied at postmortem. In 1888, Horschsprung wrote his classical description of the condition, and two years later reported 2 cases before the German Pediatric Society. Tilger reviewed the German, French and English literature in 1893 and collected 23 cases, adding one of this own proved at postmortem examination. The palpable tumor was first called attention to by Finkelstein in 1896. Persistence of the relatively thick pylorus normally present in the fifth and sixth months of embryological develop-

ment has been suggested as the cause in both the adult and infantile forms. The symptoms may be mild and escape detection, and the condition is not suspected until later in life when obstruction develops and the symptoms demand either medical or surgical intervention. Morton offers the opinion that the varying degree of hypertonicity and spasticity seen in infancy may carry over into childhood and adult life, or that the process may arise in an entirely normal person. Crohn also felt that the adult type of hypertrophy of the pylorus represented a carry-over from the infantile form, and doubted that many would be seen in adult life because of the earlier intervention and successful operation.

Oliver felt that the etiological factor in both the infantile and the adult type was the same, inasmuch as we are dealing with the same condition. Brown felt that, once established, the condition is permanent. Wallstein proved that in patients treated medically and those treated with a gastroenterostomy the tumor mass remained the same and was unaltered. Martin and Bordon believed that infantile hypertrophy may result from intermittent chronic spasm which begins in fetal life and may be caused by a preponderance of sympathetic influence or tardy development of vagal inhibitions. An excess of adrenal secretion which is a powerful stimulus to the sphincter may be a factor. It may be significant that infantile hypertrophy and peptic ulcer occur with greater frequency in males than in females. Nechles suggests the possibility of intrauterine influence whereby the amniotic fluid with its meconium, cell detritus and possibly air, may irritate the sphincter, with resultant edema, spasm and enlargement. This local trauma plus autonomic imbalance may be the causal factor. Longstanding spasticity is thought to be one of the causes by McClurg, who reports that in some of his cases nervousness alone is capable of producing spasticity of the pyloric muscle which if long continued may in turn cause hypertrophy of the spastic tissue. Raymond seems to discount the possibility of spasm, stating that spasm occurs in many muscles of the intestinal tract, *i.e.*, anus, cardia of the stomach without hypertrophy. He suggests that hypertrophy may represent a throw-back to the ancestral types, observing that in some birds the gizzard is located in the same spot as the pylorus. Vorhaus believes that hypertrophy of the pyloric muscle in the adult can be divided into two categories: (1) those with signs and symptoms of very short duration, and with no other gastrointestinal pathology, and (2) those with signs and symptoms from childhood.

Bachus divides the cases into three groups (a) those with signs and symptoms from childhood; (b) those with atypical ulcer symptoms of long duration beginning in adult life; (c) those who quickly develop signs and symptoms of pyloric obstruction in middle or late life. Dutton Thompson found that symptoms varied from one to forty-two years, with an average of ten and one-half years. Vomiting was the most common symptom. Weight loss and constipation was the next most common. Patients have a variety of complaints, soreness and fullness of the epigastrium, lump in the stomach, stomach fills quickly, epigastric heaviness, dyspepsia, post-prandial pain and night regurgitation of acid material. Objective findings noted most frequently were weight loss, and dilatation of the stomach. Occasionally, a palpable tumor in the abdomen could be felt. In the review of 60,000 roentgen examinations of the stomach Kirklin and Harris found 81 cases of hypertrophy of the pylorus which were verified at operation. In all 81 cases they found elongation of the pyloric ring with crescentic indentation of the bulbar base. Other associated findings were hyperperistalsis, gastric dilatation and retention. Store and Ruggells studied a group of abnormalities found in the pylorus and prepyloric region and concluded that practically no type of deformity was characteristic of a definite disease or lesion. They felt that hypertrophy of the pylorus had two

suggestive features: (a) decreased diameter of the orifice and failure to open; (b) elongation of the pylorus with an immediate prepyloric deformity. Burke believes that the roentgenogram is not diagnostic since malignant infiltration may produce concentric narrowing of the distal portion of the stomach similar to hypertrophy. Gastroscopy is of aid in ruling out ulceration and malignancy. Nickolof's studies of aspiration reveal no diagnostic changes. Acidity is of no great diagnostic importance as many are either hypochlorhydric or achlorhydric.

North and Johnson report on pyloric hypertrophy in the adult. They have collected 59 cases with operative or necropsy verification, adding 5 of their own cases, previously unreported. They are under the impression that pyloric hypertrophy in adults represents a persistence of the infantile form because morphologically the lesions are almost identical. A clinical history of life-long indigestion, vomiting, etc., given in some of the reported cases also lends support to this concept. Rundstum reports a number of interesting observations during what he calls the manifest stage. He observed dilatation of the stomach terminating in an abruptly smooth elongated canal. Peristaltic waves were interrupted at the constricted portions. The passage of the barium into the duodenum was prolonged from two to four times normal. In these patients, when examined later during the first year of life, the pyloric canal was wider and the outline of the barium within it showed longitudinal streaks. He interpreted the streaks as mucosal rugae which have been obliterated by edema during the stage of clinical symptoms. This stage seemed to continue from one to four years. The abnormality gradually regressed but in some cases the normal pattern was not established even as late as the thirteenth year. These observations by Rundstum show quite clearly that an organic basis may remain in unoperated patients as a potential source of trouble in adult life. In a group of 64 cases collected in a series, there was a ratio of males to females of about 3 to 1. However, in a series reported by Judd and Thompson, in 20 proved cases the sex ratio was 19 males to 1 female.

Doerma's account of gastrointestinal disease observed in the Netherlands during the Nazi occupation shows some interesting sidelights in the spasticity theory as the cause of hypertrophy of the pyloric muscles. He observed that the restricted diet mainly of coarse carbohydrate food, upon which these patients subsisted, caused various spastic diseases of the gastrointestinal tract. During this period roentgenologists observed in many instances a rigid gastric antrum in which hypertrophy of the pylorus, without ulcer or cancer, was the sole finding at operation. It was unlikely that the malnutrition accounted for this finding. Atrophy rather than hypertrophy is customary in people dying of starvation.

The roentgenologist is the one who most frequently discovers this condition in the stomach, although it may be very difficult in the adult to distinguish and differentiate it from an infiltrative type of neoplasm. Many of these patients have a gastric residue, but as a rule the residue is not as large as that encountered in annular carcinoma or stenosing ulcer. Only in very rare cases is complete gastric retention encountered. Usually the stomach contains about a 25 per cent residue in the six hours. The degree of hyperperistalsis is variable. Often the pyloric canal can be visualized due to the fact that barium passes through the duodenal bulb and there is a significant elongation of the pyloric canal. The distribution of the muscular hypertrophy determines whether or not the canal will be concentric or eccentric. On careful examination, occasionally irregularities can be seen produced by edematous rigid folds which may project into the barium column. Usually these folds do not retain the barium as is the case in pyloric ulcer. Kirklin described a large series of cases with reference to the mushroom or umbrella deformity at the base of the duodenal bulb as an almost pathognomonic sign of pyloric hypertrophy. Pendergrass de-

scribed this deformity as probably due to a prolapse of the gastric mucosa or to a pedunculated tumor. This concavity may be due both to pyloric hypertrophy and prolapse of the mucosa. Usually in the latter the defect may be varied by palpation. The use of antispasmodics is of very little value in determining whether or not the narrowing is due to pyloric hypertrophy, ulcer or spasm. The antispasmodic does not alter the narrowing in the hypertrophy of the pyloric antrum or muscle. North and Johnson do not believe that a mushroom deformity of the base of the duodenal bulb is necessarily due to pyloric hypertrophy. They report cases of pyloric ulcer in which they have found this deformity. It is often difficult to recognize this lesion at the operating table. It is necessary that the surgeon carefully palpate the pyloric antrum for any thickening or unusual deformity. If he is unable to feel any mass or thickening it is necessary that he open the pyloric antrum and palpate it, also place his examining finger in the pyloric ring, carefully inspecting the interior of the stomach. The thickness of the pyloric muscle varies and has never been clearly defined. Crusdarrel and Horwitz *et al.* agree that 0.5 cm. is an average measurement, although the range in the latter series is from 0.38 to 0.35 cm. The measurements were made on 6 specimens in which the sphincter had been sectioned. The outstanding pathological finding in this lesion is the hypertrophy of the circular layer of the muscles of the pyloric ring. The longitudinal muscle coat may also show a moderate degree of hypertrophy, and the submucosa and mucosa may be thickened by edema or by chronic inflammatory reaction. Unyielding tumor is felt in the gross specimen. It involves the distal 3 or 4 centimeters of the stomach and is thickest at the pyloroduodenal junction. The hypertrophy and hyperplasia seen microscopically seem to involve chiefly the circular muscles of the pylorus but occasionally involve the longitudinal coat also. There is a subcellular infiltration that may be found in the mucosa and submucosa, consisting of plasma cells, lymphocytes and a few neutrophils. These changes are probably due to a mild nonspecific inflammation and show no tendency to proceed to suppuration. According to North and Johnson the intensity of the inflammation or inflammatory reaction is proportional to the duration and the degree of pyloric obstruction. Regarding treatment, they believe that if the symptoms are mild and indefinite no treatment is indicated. Some patients live a great many years with very little, if any, trouble, even though their roentgenological findings arouse suspicion of a pyloric carcinoma. The fact that it is very difficult to differentiate this lesion from a carcinoma has led to the very common and relatively safe exploratory laparotomy to clarify the diagnosis.

It is well known today by most gastroenterologists that in cases of cardiospasm and megacolon a segment of the cardia or rectum appears grossly to be normal. But when these segments are examined histologically marked destruction is found in the ganglions of the main nerve plexus. Alvarez[3,4] has pointed out that in lower forms of animal life, with a smooth type of muscle, separation of the muscle from its first ganglion cell caused it to cramp down in a knot and remain that way. According to Alvarez this is the simplest possible explanation for the permanent spasm of the bowel. Narrow napkin ring-like obstructing segments similar to those found in the cardia and in the rectum have also been found in the small intestine. One would expect to find a similar loss of ganglion cells associated with congenital pyloric stenosis. It is rather peculiar, however, that there is hypertrophy of the muscle which does not occur in the cardia or rectum. In 1943, Dr. Arrigo Raia of São Paulo, Brazil, published a monograph in which he discusses the findings in 14 cases of congenital pyloric stenosis. He makes the statement that one might expect to find destruction of the nerve plexus with an absence of ganglion cells in congenital pyloric stenosis. Raia reported a case in 1943 of pyloric stenosis in

an infant, and in the specimen removed, he found destruction of the ganglion cells.

Knauer, in an article published in 1938, noted destruction of the plexus of Auerbach in cases of congenital pyloric stenosis. According to Raia infants in Brazil who suffer from pyloric stenosis are particularly likely to suffer also, as one might expect, from either cardiospasm or megacolon. Heredity seems to play some part in congenital pyloric stenosis. Alvarez further reports the findings of Iamy and his colleagues of hypertrophic pyloric stenosis in twin boys. In thirteen of fifteen pairs of monozygotic (identical) twins, both children had the disease. Reports of twenty-three pairs of dizygotic (fraternal) twins were found, and in twenty-one of these only one twin had pyloric stenosis. Iamy and his colleagues concluded that hypertrophic stenosis of the pylorus is a genotypic disease. Usually there is a concordance in monozygotic twins, while discordance is the rule in dizygotic twins. Two hundred and twelve families with 556 children were studied. Out of these 556, there were 222 children (boys) (80.6 per cent) who had pyloric stenosis. In eight of the two hundred and twelve families, 2 children had the condition and in only one family 3 children had it. Consanguineous marriages can favor the development of this condition. The authors did not think the figures proved that first-born children are particularly susceptible. The difference is not large enough to be conclusive. It has been demonstrated that a longitudinal incision through the circular muscular layer of the pylorus in hypertrophic pyloric stenosis (commonly called the Ramstedt operation) will cause hypertrophy of the pyloric circular muscle to disappear and the muscle to regain its growth and microscopic appearance within a matter of months. To explain this on a neurogenic basis would be rather difficult, as the cause of hypertrophic pyloric stenosis, except that possibly enough of the vagal fibers and fibers of the myenteric plexus have been interrupted by the incision. There is a marked diminution of the vagal stimuli which will allow restoration of the ganglia and the muscle to normal.

The number of ganglion cells and the nerve fiber tracts in the myenteric plexus for a unit area of muscle tissue increases progressively to a point just below the incisura of the stomach down through the pylorus to its junction with the duodenum. The thickness of the muscle increases in the pylorus from a point just below the incisura to the pyloroduodenal junction. This increase is mostly in the circular muscle. There is an abrupt line of demarcation in the duodenal musculature of only about half the thickness of the gastric muscle just below the incisura. This is observed in the normal stomach, duodenum and pylorus of infants and adults, and also in those of infants and adults affected by hypertrophic pyloric stenosis.

Segments of the longitudinal muscle are often found to be absent in the pylorus of normal infants, as well as in the pylorus of those infants who have hypertrophic pyloric stenosis. There is a fibrous block between the circular muscle of the pylorus and that of the duodenum, whereas the longitudinal muscle is continuous in muscle is continous in two segments. Hypertrophy of the circular muscle in infants with hypertrophic pyloric stenosis will persist into adult life if gastroenterostomy alone is performed in infancy. Spaulding *et al.* came to the final conclusion, "our findings then support the neurogenic theory of the etiology of hypertrophic pyloric stenosis."

ANTRAL GASTRITIS

Antral gastritis is of great importance when one considers the difficulty of diagnosis. It is not a common disease and is probably due to some inflammation of the antral portion of the stomach of unknown etiology. The process probably begins in the mucosa and often involves the submucosa and may even extend into the muscularis. It is frequently deceiving and is quite similar roentgenologically to a malignancy in the same region. Often the roentgenological and the clinical diagnoses are made with great

difficulty. Most of the cases of antral gastritis have been reported in males between the ages of forty and seventy. Some writers, however, have reported an equal distribution between the sexes. Hyperacidity is a frequent finding in the majority of patients.

Benedict[11] is of the opinion that gastritis is the most common disease of the stomach occurring not only independently but in association with ulcer and cancer. At the Massachusetts General Hospital in the Department of Radiology a diagnosis of gastritis is made when there is thickening of the rugae and hypersecretion.

Schatzki[93] states that the rugae are wider, higher and more rigid than normal folds and they do not disappear on pressure. The importance of increased gastric secretion is also stressed and small round areas of decreased density due to small particles of mucus are present. The gastroscopist has an unquestioned superiority over the roentgenologist in differentiating the different types of gastritis. In probably 90 per cent of patients with superficial or acute gastritis, often due to alcoholism, seasickness, airsickness, dietary upsets and indigestion, the condition is of very short duration and does not require roentgenologic or gastroscopic examination. In 1,300 patients examined gastroscopically by Benedict,[12] chronic or hypertrophic gastritis without other gastric or duodenal pathology was found in 117 cases, or 9 per cent. Pain, vomiting, bleeding, sour eructations and heartburn were frequent symptoms. Pain may simulate ulcer pain. Very severe hemorrhages do occur from gastritis alone. Forty-two per cent of the 117 cases had bleeding. In only one-third of the 117 cases was a roentgen diagnosis of gastritis mentioned. In Benedict's series, 16 cases proved by sections to have only gastritis when surgically explored. Twelve were explored due to the possibility of cancer, 3 because of hemorrhage, and one because a jejunal ulcer was suspected. The gastroscope was more accurate than the roentgen ray 9 times, and twice the roentgen examination was correct. In 5 cases both methods failed to make a diagnosis. Some authors feel that an excessive use of coffee, alcohol or tobacco may be the cause of antral gastritis in some patients. On the other hand, a few patients have been reported who did not use tobacco or alcohol and only occasionally used coffee. Patients are advised by most internists to abstain from the use of coffee, alcohol and tobacco and usually there is a general improvement. Most patients showed an improvement if placed on an ulcer regimen. Gastritis may be confined to the pyloric antrum but frequently involves the entire stomach.

Means of Diagnosis. The gastroscopist and roentgenologist should work in close cooperation with the internist in trying to arrive at a diagnosis of antral gastritis. Even the closest cooperation often leaves the diagnosis in doubt. Arendt[7] emphasizes the importance of gastric analysis in arriving at a differential diagnosis. He advises the fractionated aspiration rather than the Ewald meal after a cup of tea and a piece of buttered toast has been given. He believes that in chronic hypertrophic antral gastritis a high incidence of hyperchlorhydria is present, definitely more frequent than in pangastritis. The hyperchlorhydria may be of value in a differential diagnosis of carcinoma. Subacidity and anacidity are occasionally found in antral gastritis which may be due to mucous secretions, or, as he states, insufficiency of the secreting glands in the later stages of the inflammation.

A number of years ago gastritis was thought to be a very uncommon disease. In fact, Alvarez in his book, "On the Mechanics of the Digestive Tract" second edition, speaks of its infrequency. He quotes Beitzke[9] and Hirsch[54] to substantiate his belief. In his book Alvarez states that "Gastritis is found only in the neighborhood of large ulcers, carcinomatous or benign." To quote further, "it would seem now to be a poor diagnosis to make even in alcoholics since Hirsch in 1916 has shown that true inflammation and inflammatory changes

are hard to demonstrate in the stomach of patients dying from delirium tremens." Hirsch in a recent conversation still maintains that gastritis is a very rare disease. Berg[15] reports that in 5,520 roentgen examinations of the stomach during thirty months, 139 cases were diagnosed roentgenologically as antral gastritis. Eighty of the 139 cases had antral gastritis associated with duodenal ulcer, duodenitis, gastric ulcer or gastroenterostomy. Fifty-nine of the 139 cases had antral gastritis without other demonstrable lesions in the stomach or duodenal bulb. Nineteen of the 59 cases showed other findings, such as cholecystitus, non-functioning gallbladder and herniation of the gastric mucosa into the duodenal bulb.

The roentgen diagnosis of antral gastritis is based on a close study of the mucosal pattern of the empty stomach. The following points are of value in the differential diagnosis: Granular appearance, irregular translucencies, abrupt changes in direction and break in the continuity of a fold may be due to either carcinoma of the antrum or chronic hypertrophic gastritis. A violent break in the contour usually means malignancy; rugosity, stiffening and widening of the folds with the pattern preserved suggest gastritis. Grape-like polyps in the pyloric antrum may be neoplastic or inflammatory and a histological study is necessary for differentiation. Wart-like elevations may be widespread and often arise from an atrophic mucosa. The roentgenograms may show a cobblestone appearance with streaks and valleys of barium between and around the elevations.

In the presence of a deformed antrum and early openings of the pylorus, carcinoma must be the first consideration; usually delayed opening accompanies antral gastritis. Schindler's gastroscopic observations are of value in the differentiation between polypoid, hypertrophic antral gastritis and carcinoma of the antrum; namely regularity and granular appearance suggest gastritis, variability and irregularity suggest carcinoma. If it is not possible to differentiate between a malignant and non-malignant condition, operation is imperative after a limited period of medication and dietary management.

Classification of Gastritis. The general classification may be acute or chronic. It may be further subdivided according to the work of Magnus, which is based on histological studies of 20 normal stomachs removed fifteen minutes after death and of 100 partial gastrectomy specimens, as follows:

1. Acute phlegmonous bacterial gastritis.
2. Acute corrosive gastritis.
3. Idiopathic non-inflammatory atrophic gastritis with Addison's anemia.
4. Simple inflammatory gastritis, acute and chronic.

The acute variety may be diffuse or localized to the pyloric antrum. All types, diffuse and localized, may become chronic. At this stage the term chronic atrophic inflammatory gastritis may be used. Histopathologically the disease is characterized by erosions, exudate in the gastric tips and infiltration of the mucosa. Magnus regards gastritis as a common lesion, usually quiescent and harmless, but occasionally it may progress into peptic ulcerations. Schindler's classification is based on his extensive gastroscopic observations, and is as follows:

Acute gastritis which he divides further into four types:

(A) Acute—simple exogenous gastritis due to irritating foods or other substances ingested by the subject. Gastroscopic examination reveals hyperemia of the mucosa, excess of mucus and occasional submucosal hemorrhages.

(B) Acute corrosive gastritis which follows the ingestion of corrosive fluid.

(C) Acute infectious and toxic gastritis in the course of certain diseases, such as typhoid fever, scarlet fever and diptheria.

(D) Acute suppurative phlegmonous gastritis. This type of gastritis does not lend itself to either gastroscopic or roent-

genologic examination. Chronic gastritis is a common type diagnosis, and usually covers many gastric symptoms which cannot be placed in any definite category. The etiology is unknown and it is rare that a biopsy is possible. The roentgen findings in this type of gastritis are not very reliable. Gastroscopic examination offers the best chance for diagnosis. It is of value in sorting out the different varieties of gastritis and is probably the only real advance over the method of ocular inspection since Beaumont in 1838, which was later elaborated on by Wolf in this century.

Probably the most commonly accepted classification based on the gastroscopic appearance of the surface of the mucosa is that of Schindler, and is as follows:

1. Chronic superficial gastritis.
2. Chronic atrophic gastritis.
3. Chronic hypertrophic gastritis.
4. Chronic gastritis of the postoperative stomach.

In chronic superficial gastritis, the gastroscope reveals reddening of the mucous membrane, edema, exudation and hemorrhages into and erosion of the mucosa. Schindler has described cases with infiltration of the subepithelial mucosa by plasma cells, lymphocytes and granulocytes and cloudy swelling of the epithelial cells. The histopathology is not established in these cases. The etiology is unknown, but irritants, food, alcohol, nicotine and hot fluids have been accused. Gastric analysis may reveal a normal degree of acidity; there may be too much, too little, or anacidity may be present. These patients usually make a good recovery if treated energetically. In this type of case the roentgen examination fails to show any significant change.

Chronic atrophic gastritis may be secondary to an untreated superficial gastritis. It is frequently present in Addison's anemia and in gastric carcinoma. Some writers believe that it often precedes the development of carcinoma. The gastroscopic examination may reveal gray or green thin patches on the mucosa. In the very advanced stages the blood vessels may become visible. In some of the advanced cases peptic glands may entirely disappear. There may be infiltration of round cells, plasma cells and fibroblasts. Complete achlorhydria is usually present. Veld and Schindler agree that the roentgen appearance is so variable as to be without serious diagnostic value. In cases described as achylia gastrica, reported by Veld, usually there is a normal relief pattern and a few have narrow rugae, and others have very coarse rugae. Some of the cases described by Schindler showed a marked atrophy. The roentgen pattern was very coarse. The rugae were of normal size gastroscopically. This seems to confirm the view that the pattern seen roentgenographically is dependent on the autoplastic mechanism of the submucosa and not on the epithelial layer. In far advanced cases in which there is much submucosal fibrosis the roentgen appearance is often that of a simple type of leather bottle stomach.

Chronic hypertrophic gastritis involves principally the body of the stomach and rarely the pyloric antrum. The changes may be diffuse or patchy. Gastroscopically the signs are a velvety swollen spongy appearance with so-called highlights. Later small nodules develop which gradually increase in size reaching a mammillated or polypoid appearance. There may be superficial erosions which usually heal quickly. These may cause hemorrhages which may be slight or severe. Histopathological changes are proliferation of the entire mucous membrane, including the peptic glands and the cellular infiltration of the interstitial tissue of the mucosa. Here, again, very little is known of the etiology. Schindler believes, after long years of gastroscopic study, that the development of peptic ulcer following or accompanying gastritis is a rarity. He also does not believe that chronic hypertrophic gastritis predisposes to carcinoma. There is still much disagreement between the gastroscopist and the roentgenologist regarding the value of the two methods of

diagnosis. Gastroscopic examination usually reveals surface changes, gives very little information regarding the underlying structures, that is, the submucosa, and so forth, whereas the roentgenological signs are often due to changes below the mucosa. It is also stated by a number of roentgenologists that the inflation of the stomach for successful gastroscopic examination often deletes the mucosal folds which are seen roentgenoscopically and roentgenographically. Chronic hypertrophic gastritis is a common roentgenological diagnosis and is based on the coarse mucosal pattern seen in the mucosal relief picture but seldom proved histologically. These changes which are described by roentgenologists are often misleading. In many cases a very coarse mucosal pattern shows a normal gastroscopic appearance.

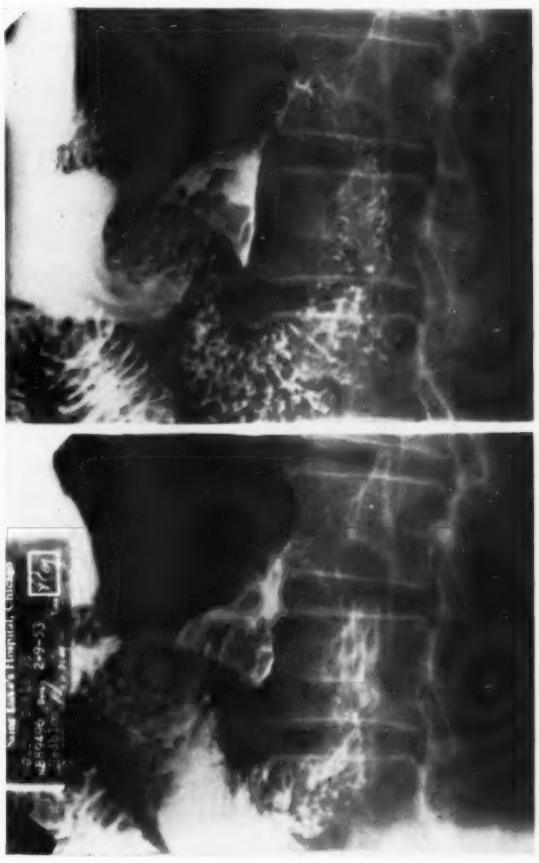

Fig. 3. Patient O. G. Prolapse of gastric mucosa; proved.

The gastroscopist believes that the only roentgen signs of value are the nodular or corncob pattern, which is rarely seen. Gutzeit, Maley and Veld, Henning, Schindler and Moutier claim that very little reliance should be placed on roentgen findings unless the changes in the roentgenogram are very prominent such as in the giant type of gastritis, seen usually on the greater curvature in the fundus of the stomach. The changes, however, seen in antral gastritis roentgenologically do not conform to the changes that appear in the giant type of chronic gastritis commonly found along the greater curvature of the pars media and pars cardia. Often there are no mucosal folds outlined roentgenoscopically or roentgenographically. Frequently it is impossible to study the mucosal pattern due to the narrowing and spasticity of the pyloric antrum. This is one of the important diagnostic points in differentiating antral gastritis from either a pyloric hypertrophy or a scirrhous type or infiltrating type, superficial in character, carcinoma.

In our experience antral gastritis is an uncommon disease, especially if the diagnosis is based on histopathological findings. Very few cases come to operation; consequently, only in rare instances can we prove our roentgenological diagnosis.

Prolapse of the Gastric Mucosa (Fig. 3 and 4)

Prolapse of the gastric mucosa into the duodenal bulb is not an unusual finding and is encountered quite often in a busy roentgen department. The gastric mucosa may prolapse through the pylorus and into the duodenal bulb without any other co-existing pathology. Accompanying prolapse of the mucosa may be an inflammatory process causing bleeding of the redundant mucous membrane. This may on occasion warrant surgical intervention. The diagnosis is usually made by roentgenographic and roentgenoscopic studies. The roentgen findings are usually a cauliflower-like defect often described as an umbrella-shaped mushroom. There is usually a deformity

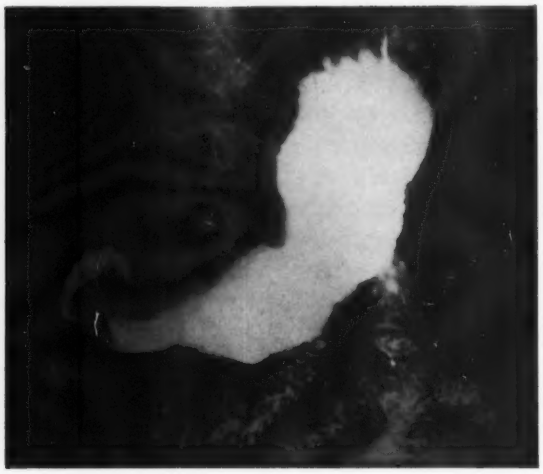

Fig. 4. Patient O. G. Prolapse of the gastric mucosa.

seen at the base of the duodenal bulb, and the folds of the gastric mucosa are visualized in the pyloric antrum and in the duodenal bulb. Often there is a definite widening of the pyloric canal, especially when the patient is in the prone or in the George position. The prolapsed mucosa may cause a smooth crescent-shaped defect at the base of the duodenal bulb. A smooth crescent-shaped defect of the duodenal bulb due to pressure or overlap of the pyloric valve may be confused with prolapse of the mucosa. A smooth crescent-shaped defect of the duodenal bulb may occur in both conditions. Prolapse of the mucosa must also be differentiated from pedunculated polyps and indurations associated with an ulcer, or duodenal inflammatory changes. Prolapse of the gastric mucosa is usually seen in middle aged persons who have upper abdominal pain not relieved by medical ulcer regimen. Nausea and vomiting may be the most important manifestation. By changing position, the patient may sometimes alleviate his night pain.

In a series of cases hemorrhage occurred in about 25 per cent of the patients and occasionally a severe anemia with tarry stools or hematemesis occurred. As a rule, surgery is not indicated and most physicians and surgeons are rather reluctant to advise surgery. Only in the presence of complications or other accompanying pathology, such as gastric ulcer or very severe symptoms, does it seem that surgery is indicated or should be carried out. If surgery is indicated, it is probable that gastric resection is the procedure of choice. About 50 per cent of patients with uncomplicated gastric mucosal prolapse were improved on medical management. The great majority of patients on whom operation is indicated are greatly benefited by the operation. In rare cases the gastric mucosa becomes loose and very redundant, and may at times hang in folds. When this is the case the mucosa herniates through the pylorus into the duodenal bulb and may simulate a tumor, such as a small polypoid growth.

Rees[85] found 9 proved cases of prolapse of the gastric mucosa without tumor formation reported in the literature. He added 4 more cases of his own, 3 of which were proved at operation. The fourth had not been operated on at the time of the report. The chief symptom in his series of cases was periodic distress in the upper portion of the abdomen characterized by a sensation of excessive fullness and cramp-like pain in the right upper quadrant, especially after eating. Vomiting may occur in these patients, with or without demonstrable obstruction. The patients may have long intervals in which they are free from symptoms.

Usually the condition seems to progress and the mucosa becomes more redundant. The roentgen findings with the barium meal reveal a negative shadow containing a central opaque streak in the duodenum. Usually the patient's stomach is free of barium at the end of six hours and often there is a slight hypertrophy of the rugae demonstrable. In none of Rees' 3 patients operated on was there evidence of polypoid degeneration, but in all a constriction of the pyloric ring was found and the mucosa of the antrum was thickened, and showed oblique folds that could be pushed through the pyloric ring.

Extragastric pathology (Fig. 5) is of

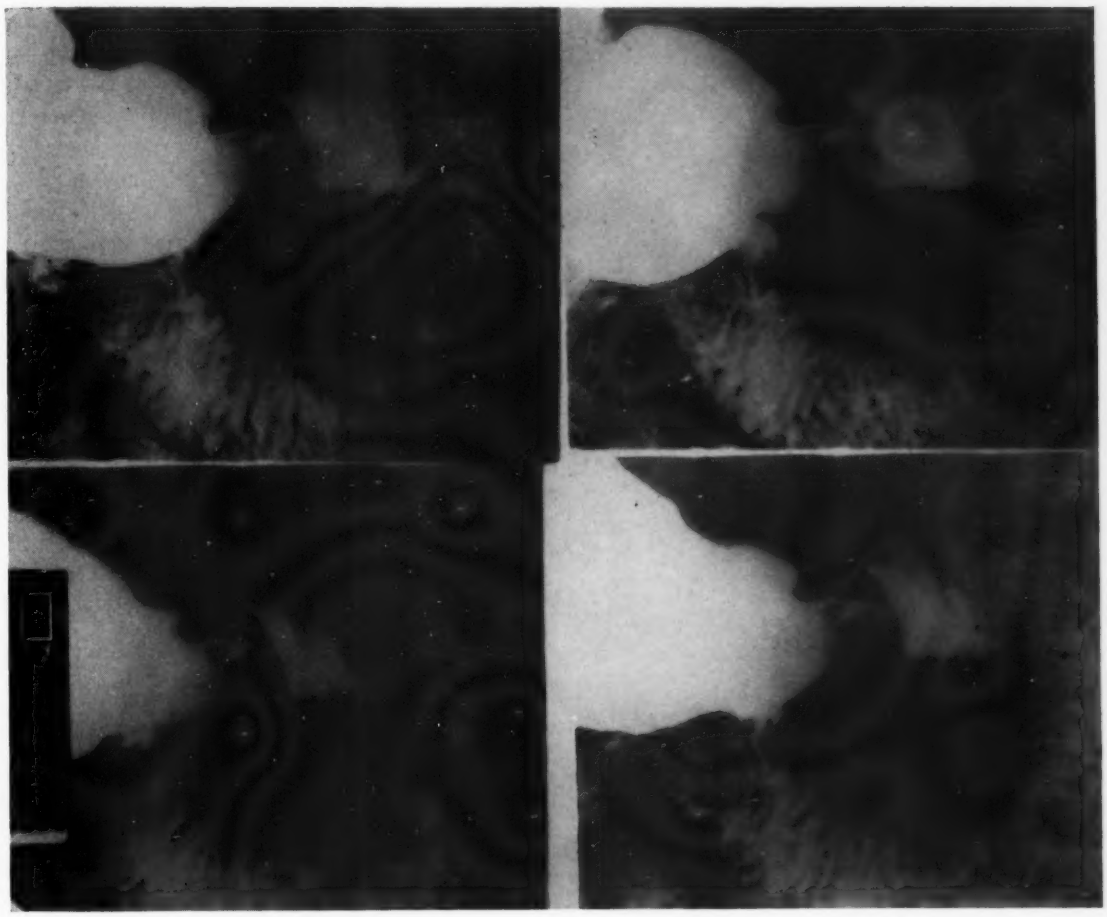

Fig. 5. Patient D. P. Pyloric narrowing due to adhesions; proved at operation.

great importance and often makes the diagnosis of gastric lesions very difficult. Perigastric adhesions and bands involving the pyloric antrum are not uncommon and it is often impossible to make a correct preoperative differential diagnosis. Intramural gastric tumors, far advanced infiltrations, and benign ulcerations are more often readily diagnosed from a clinical and roentgenological standpoint than perigastric adhesions and bands involving the pyloric portion of the stomach. Many of these deformities are roentgenologically compatible with defects produced by malignant gastric pathology. Deformities involving the pyloric antrum due to congenital bands and adhesions were described as early as 1914 by M. L. Harris. At that time he described deformities of the duodenum resulting from anomalous congenital bands. It was his opinion that the adhesive bands were the remains of the anterior mesogastrium resulting from faulty embryonic development. Pressure was exerted on the duodenum and pyloric antrum due to ligamentous folds. If the bands are confined to the second and third parts of the duodenum the pyloric antrum is involved only indirectly causing dilatation of the pyloric portion of the stomach due to pressure on the second and third parts of the duodenum.

In 1922, A. S. Taylor reported a number of cases on which he had operated for symptoms arising from anomalous abdominal membranes. He felt that these abdominal membranes were developed during fetal life from atypical peritoneal fusion,

FIG. 6. Patient A. B. Deformity due to falciform ligament; confirmed at operation.

later modified by pathological changes probably resulting from continued traction or possibly low grade inflammation. The patients may develop obstructive symptoms, digestive disturbances, nutritional changes, and nervous debility and often complain of epigastric tenderness.

Previously we have reported a number of patients with defects due to perigastric adhesions and bands causing deformities of the pyloric antrum of the stomach, with absence of surgical and pathological findings. Our discussion at that time was limited to adhesions and bands deforming the pyloric antrum which we described as the distal 2.5 centimeters of the stomach adjacent to the pyloric sphincter. It is in this region that benign gastric deformities may closely resemble intramural gastric malignancies. Congenital anomalies and inflammatory adhesions are not infrequent. In one of our cases, a male, a deformity was found involving the pyloric antrum causing a funnel-shaped narrowing (Fig. 6). This was discovered during the roentgen examination and was thought to be due to malignancy. At operation an adhesive band which was continuous with the lower part of the liver and the falciform ligament was found surrounding the pyloric end of the stomach about 1 inch proximal to the pyloric sphincter. At operation the band shelled off the anterior wall of the stomach and after division revealed a normal stomach beneath. Nothing suggestive of malignancy was found. The roentgen examination was very suggestive of an intraluminal malignancy. The narrowing of the pyloric antrum was constant at several examinations and was not affected by antispasmodics. This portion of the stomach seemed to be rigid on palpation and, due to the encirclement, no peristaltic waves were evident. The encirclement by the adhesive bands also caused a marked distortion of the gastric rugae. Again recalling the normal anatomy of the pyloric antrum and the gastric attachments, one finds that the lesser curvature is covered by peritoneum giving rise to the two layers of gastrohepatic omentum. Its two components, the gastrohepatic and duodenohepatic ligaments, pass upward to attach about the hepatic portal. The right lateral margin is reflected around the hepatic artery, bile ducts and portal vein, turning backward to form a free folded edge of the peritoneum. As it winds about the portal vessels it becomes the anterior boundary of the foramen of Winslow. On the greater curvature, through reflection of the peritoneum around the pylorus, it forms the attachment of two layers of the greater omentum. It forms the gastrocolic ligament uniting the stomach and transverse colon. Normally these gastric ligaments allow the stomach considerable mobility and interfere in no way with the passage of peristaltic waves over the gastric walls. Roentgenoscopically the prepyloric portion of the stomach and duodenum is relatively more fixed than the pars media, but, even so, may be moved several centimeters by the palpating hand. Fixation of the stomach is highly sugges-

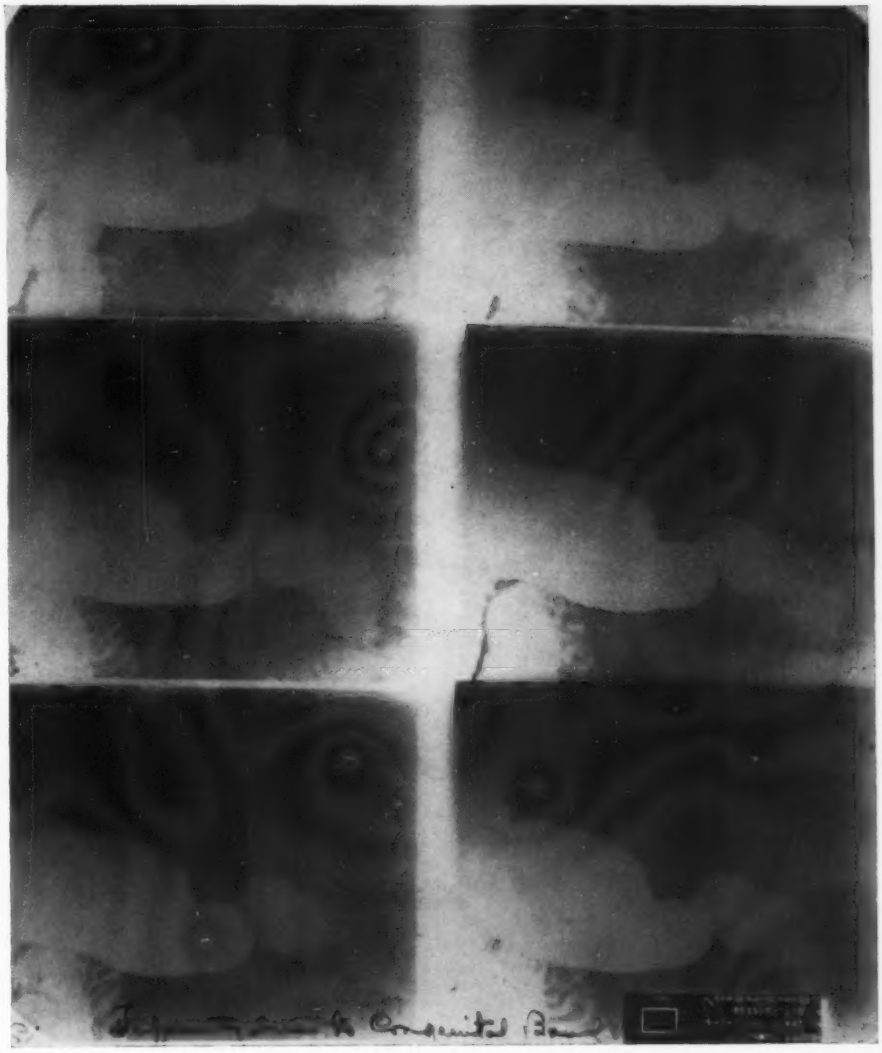

Fig. 7. Patient S. B. Deformity lesser curvature pyloric antrum due to congenital band; proved at operation.

tive of extragastric pathology, but when fixation is associated with a filling defect deforming either curvature, the extension of gastric pathology into the surrounding tissue is highly probable. Such a finding, together with rugal disturbance and inhibition of peristaltic waves, usually leads to surgical explorations. The free edge of the lesser omentum may be thickened and short. Included in this thickened lesser omentum may be the gastrohepatic ligament.

S.B., a white male, aged fifty-two, complaining of gas and heart pain, was examined roentgenologically and a deformity was found on the lesser curvature of the stomach near the pyloric sphincter (Fig. 7). The deformity was constant. Peristaltic waves were absent along this portion of the lesser curvature and the filling defect was rather smooth and was present after administration of antispasmodics. We thought after repeated examinations that we were dealing with an organic filling defect and advised operation. The patient was operated on and a deformity was found involving the lesser curvature of the stomach approximately 2 cm. from the pyloric sphincter fixing this portion of the stomach and elevating it, causing a marked angulation in such a manner as to depress the lesser curvature

distal to its attachment. The deformity, at operation, was thought to be due to the gastrohepatic ligament attachment. The stomach was inspected, opened, and the mucosa was found to be normal.

Narrowing of the pyloric antrum may be due to adhesions between the greater omentum and the pyloric antrum with displacement of the greater curvature. We have had one such case which was explored and the stomach was found to be normal except for a scar at the point of adherence of the greater omentum. The close relationship of the gallbladder to the duodenal bulb and pylorus also complicates pyloric antrum pathology. Irregular narrowing, fixation and a rigid pyloric antrum may follow or accompany gallbladder pathology. On roentgenoscopic examination the peristaltic waves may end abruptly at the area of narrowing. In one of our patients the lumen of the stomach was narrowed to less than 1 centimter in diameter and the rugae could not be outlined. The patient had had a previous cholecystectomy. On exploration the entire right upper quadrant was obliterated by dense fibrous adhesions extending like a partition from the old gallbladder scar to the posterior abdominal wall, contracting and absolutely fixing the pyloric end of the stomach. It was necessary to do an extensive dissection of the pyloric antrum of the stomach from the first part of the duodenum before the adhesions could be divided and the stomach freed. The stomach was incised and no intramural lesion could be found. Following the operation the patient was again roentgenoscoped and there was still a distortion of the pyloric antrum, but the rugae previously not seen could now be outlined.

Perigastric adhesions in the region of the pylorus may also arise from pathology within the stomach. In 1937 we examined a patient and found a small ulcer at the base of the duodenal bulb. Several years later the same patient was found to have developed a narrowed irregular pyloric antrum with inhibition of the peristaltic waves and with rugal disturbance. This patient was operated on and found to have developed a marked fibrosis surrounding the pyloric antrum originating at the site of the old ulcer with a marked deformity involving the prepyloric area. At operation there were no nodules present. The liver and gallbladder were normal and there were no indications of a malignancy or metastases. A posterior gastroenterostomy was performed. It seems that most of the patients with perigastric lesions are males over fifty years of age. This may be due to continued traction and low grade inflammation and changes in the habitus of the patient. In all the patients we have observed with perigastric adhesions there has been a complaint of vague epigastric distress and weakness. All of the patients complained of distress after meals; only one had a history of vomiting. Weight loss was a variable factor. Most of the symptoms seem to be caused by strong peristaltic waves necessary to force the food past the point of narrowing. Of great importance from a clinical standpoint is the fact that every one of the patients that we have seen had signs of gastrointestinal bleeding. All but one had an achlorhydria. Only one patient that we have seen had known pathology to account for the bleeding. This patient had a duodenal ulcer. Tarry stools are a common finding, and a 4 plus benzidine reaction on a meat-free, gravy and iron-free diet is the rule rather than the exception. Gastroscopic study is not of great value in arriving at a diagnosis as this area is difficult to examine. The differential diagnostic features of lesions of the pyloric antrum have been fully described, but there is still considerable uncertainty as to the exact cause of these filling defects. In other words, it is often impossible to arrive at a definite diagnosis even after thorough roentgenographic examination, gastroscopic examination and a careful history and clinical examination.

The difficulty of differential diagnosis of lesions of the pyloric antrum is exemplified by a patient whom we examined in 1941.

A male, R.C., aged fifty-one, complained of pain varying from dull to intense in the upper abdomen, with epigastric distress following meals. The patient occasionally vomited and failed to obtain relief from alkaline powders. Gastric symptoms dated back to 1940, at which time he first experienced epigastric distress, discomfort, bloating and belching. Other than these symptoms he felt quite well, showing no lassitude, loss of weight or weakness. He had been examined five days previous to admission to our hospital by Dr. James T. Case and a diagnosis of an organic lesion of the pyloric antrum was made, the exact nature of which was not stated. Dr. Case, however, ventured an opinion that he thought it might be inflammatory. The blood count was within normal limits and the gastric analysis showed 56° of free hydrochloric acid, and total acidity of 68°. Blood Kahn tests were negative on two occasions. Two stool examinations were negative for blood and amebae. Blood chemistry and sedimentation rate were within normal limits. Chest examination was normal. Graham-Cole study showed a normal functioning gallbladder with no stones. Electrocardiographic examination was normal.

We examined this patient on January 2, 1942, after the administration of atropine sulfate grains 1/100 by mouth, twenty-four, twelve and two hours before examination. Roentgenoscopic and roentgenographic examinations at this time revealed a circumscribed filling defect involving the walls of the pars pylorica (Fig. 8). There was no large intraluminal mass. The lines of demarcation were rather sharp and the rugae appeared to be irregular. In the pars cardiaca and pars media the rugae appeared distorted and thickened. On roentgenoscopic and roentgenographic examination the pyloric defect was constant and the peristaltic waves did not traverse this area. Comparison of our films with those of Dr. Case leaves no doubt as to constancy of the lesion. The duodenal bulb was within normal limits. Our diagnosis was a filling defect involving the pars pylorica which should be considered malignant until proved otherwise.

Gastroscopic examination was done but offered no additional information as the pyloric antrum could not be seen satisfactorily. The body of the stomach was smooth and the mucosa normal. Projections beyond the angularis on the lesser curvature were thought to be thickened folds.

Fig. 8. Patient R. C. Pyloric antrum deformity; no pathology at operation or on sections.

At operation, the stomach and duodenum appeared externally normal. Nothing was found in the nature of induration, nodules or glands in the stomach. The gallbladder, liver and surrounding regions were normal to inspection and palpation. In view of the roentgen findings, an incision was made 1 inch above the pylorus on the anterior wall of the stomach. A section was removed for gross and microscopic examination. The lining of the stomach and the first part of the duodenum were inspected, palpated and found normal. Microscopic examination of the biopsy specimen by Dr. E. F. Hirsch showed normal gastric tissue with no evidence of disease.

Following the operation the patient was later examined on numerous occasions, roentgenographically and roentgenoscopically. Whereas his general health seemed to be quite satisfactory the deformity involving the pars pylorica still remained. There was one interesting observation made by Dr. S. W. McArthur during the operative procedure, namely palpation of the pyloric antrum gave a definite impression of induration of the entire region. After a short pause, with the pars pylorica still between the thumb and index finger, this impression vanished entirely. On further palpation and visual inspection he observed an extraordinary spasm involving the stomach when first touched, followed by relaxation with nor-

mal consistency to palpation. During the entire operative procedure any traction on the stomach or palpation of the splenic area produced a profound regional gastrospasm with prompt cessation of respiration. In view of the profound gastric spasm this patient exhibited under general anesthesia, one would expect very little affect from antispasmodics given prior to the second roentgen examination.

Antispasmodics seem to offer very little in the way of differentiating spasm from organic lesions. In most of the lesions we have seen atropine or belladonna causes no change in the spasm or the narrowing. The antispasmodic action of the drug is supposed to be due to its affect upon the intrinsic nerve supply of the stomach which supplies the outer longitudinal and intercircular muscular layers. Vagus stimulation activates the stomach, whereas sympathetic stimulation relaxes it. According to the theory advanced by Forssell,[43] gastric mucosa can adapt its rugae in response to the stimulus of food, as a local autonomic response, not dependent upon the distant nerve control. Gastrospasm is usually thought of as intrinsic due to gastric pathology, or extragastric due to reflex or other stimulation of the extrinsic nerve. In the previously reported case, no pathological condition could be demonstrated, and we feel that the changes may have been due to local autonomic response. The possibility of muscular mucosal spasm must be thought of in all of these narrowed and deformed pyloric antra. One should seek diligently to find the cause of the deformity.

Even though a number of the patients we have seen have been operated upon and no pathology has been demonstrated, we still believe that there is some underlying cause of the narrowing. Certainly these deformities are not present in a normal stomach. Again, I must reiterate that all of the patients we have seen with pyloric deformities due either to perigastric adhesions or filling defects simulating carcinoma in which no pathology could be found in the stomach, were males over or in the neighborhood of fifty years of age.

Another male patient with a persistent filling defect after medical management and antispasmodics was examined roentgenologically because his appetite was poor. He complained of distress and bloating after eating. He was relieved when he belched lying in the recumbent position. At times he experienced faintness and nausea. There was no history of vomiting. The laboratory findings were essentially negative. Wassermann and Kahn reactions were negative.

Roentgen examination of the stomach on August 5, 1940, revealed a definite narrowing of the pyloric antrum for a distance of 3 cm. The rugae appeared distorted and partially destroyed. This filling defect was constant during roentgenoscopic and serial roentgenographic examination. The stomach was hyperactive but the filling defect was clearly demonstrated. The pyloric antrum was spastic and no barium passed through for about fifteen minutes. Reexamination was advised after administration of antispasmodics and two days later the examination was repeated. The pyloric antrum was again found narrowed and spastic. On some of the films, the rugae appeared within normal limits. The possibility of a hypertrophic pylorus with spasm was considered.

The patient was examined again on September 10, 1940, and practically the same findings existed. The peristaltic wave this time extended downward to the pars pylorica and then ended abruptly. The roentgen diagnosis was organic filling defect, probably malignant, but hypertrophy of the pyloric muscle had to be considered.

The patient was operated on by Dr. G. V. Pontius. It was his impression on palpation that the pyloric antrum was thickened. The stomach was opened and the mucosa revealed hypertrophic rugae, but no definite mass could be distinguished. There was no evidence of any enlargement of the lymphatic nodes. Partial resection was decided upon and the distal fourth of the stomach was removed. A gastrojejunal anastomosis was carried out. The pathology report was as follows: "No rugal or pyloric hypertrophy could be seen in the resected portion of the stomach or duodenum." Microscopically serial sections of the entire resected portion revealed no invasive epithelial growth or inflammatory process (Dr. E. F. Hirsch).

Here again, the surgeon may have felt and seen the gastric spasm which he interpreted as pyloric and rugal hypertrophy. On the basis of Forssell's work such a spasm may be postu-

lated as involving the muscularis mucosa. Belladonna or any of its derivatives would not be expected to relieve such a spasm. We again examined the patient on January 29, 1942, approximately one year after gastric resection. The stoma at the site of anastomosis seemed to be functioning satisfactorily. The remaining portion of the stomach was found to be normal except for rather prominent gastric rugae.

The lesions we have just described are not of the medullary type. We were unable to identify an intraluminal mass in any of these cases. This makes the diagnosis more difficult. Large intramural or medullary masses are not difficult to identify. These narrowed infiltrates are the lesions which offer the greatest difficulty in arriving at a diagnosis. In these cases just reported the changes appear to be quite superficial. In all of the cases both curvatures of the pyloric antrum were irregular and the area was sharply demarcated. Peristaltic waves were absent in the affected areas. Palpation gave one the impression of rigidity with loss of pliability. It was impossible on palpation to expand the pyloric antrum even when using force. In the two cases which showed no pathology at operation it is very difficult to accept that deformities of such an extent could be due to spasm alone. Possibly there were lesions high up in the stomach which we and the surgeon failed to identify, and the pathologist did not have the opportunity to examine due to the fact that only part of the stomach was removed. The gastric behavior both roentgenoscopically and roentgenographically was not that of a normal stomach. Spasm may be due to an overstimulated autonomic system, but the deformity caused by spasm is usually smooth.

CARCINOMA OF THE PYLORIC ANTRUM (FIG. 9 AND 10)

According to most investigators about 75 per cent of the carcinomas of the stomach are located in the pyloric antrum distal to the incisura angularis. In a report of 205 cases by Golden, 75 per cent of the lesions

FIG. 9. Patient W. R. Cancer of the pyloric antrum; resected and proved microscopically.

were located in the pyloric antrum; 20 per cent median and 5 per cent in the fundus. It seems to be a well accepted fact that most organic lesions of the stomach are due to carcinoma. In Stout's diagram showing the location of 120 cancers in 120 resected stomach specimens, a majority of the lesions were located in the pyloric antrum. Holmes and Hampton,[55] in a study of the material from the Massachusetts General Hospital, found that the relative proportion of a benign to malignant prepyloric ulceration is approximately one to twelve. Willis, in his textbook "Pathology of Tumours," reports 1,006 patients with tumors of the stomach. He uses the figures of Stewart, Poscharissky, and Willis. Four hundred and seventy-four of the 1,006 tumors were in the pyloric antrum, or 47 per cent; 262 tumors were located on the lesser curvature of the stomach, or 26 per cent; there were 103 located in the cardia, or 10 per cent; in the rest of the stomach there were 167 tumors, or 17 per cent. These figures are somewhat lower than most investigators report as to the frequency of car-

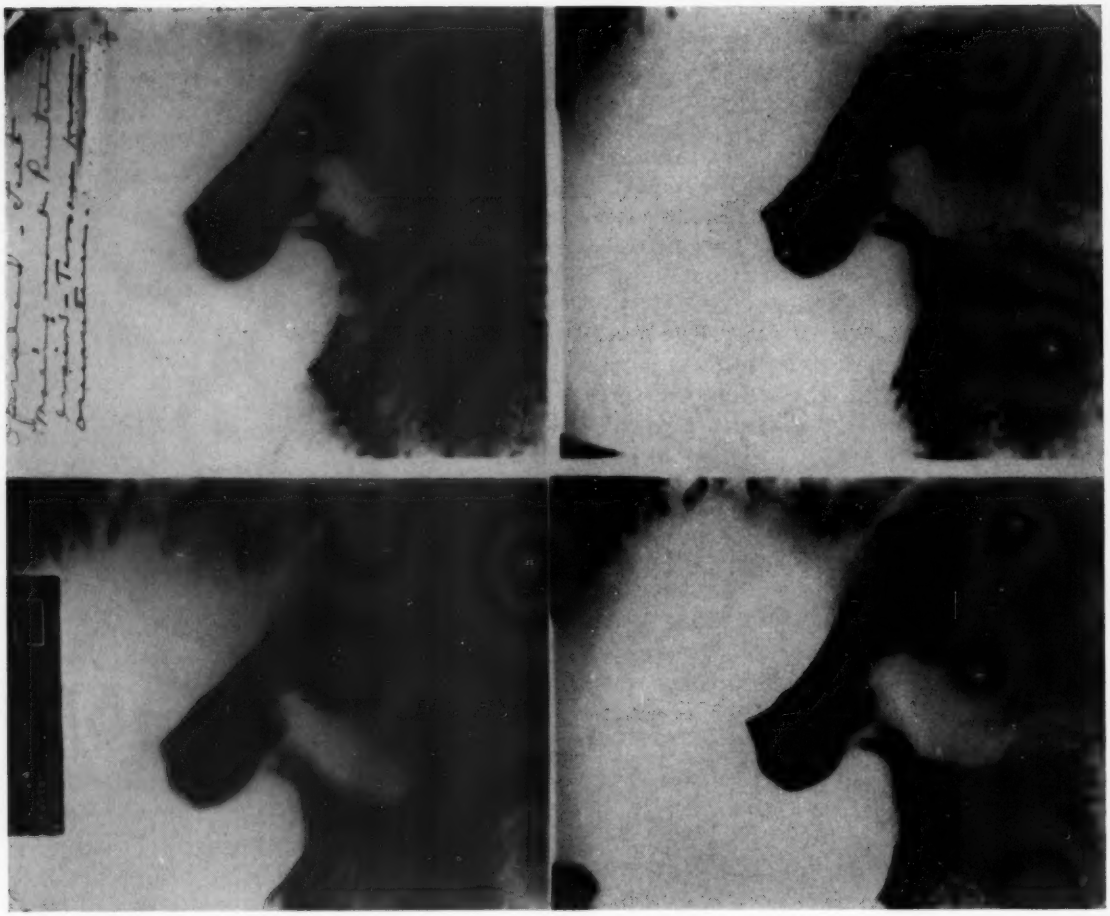

FIG. 10. Patient H. K. Cancer of the stomach with metastasis to greater omentum; proved at operation.

cinoma in the pyloric antrum.

With our modern roentgenologic technique it has become rather easy to demonstrate ulcerative lesions in the pyloric antrum. While we can usually identify lesions of the pyloric antrum, when present, it is still very difficult to make a differential diagnosis. In fact, our ability to diagnose has not improved in proportion to our mechanical means of identifying the lesions. The pyloric antrum is particularly important because a majority of all gastric lesions occur in this portion of the stomach. It is an accepted fact that it is often impossible to differentiate benign and malignant lesions in the pyloric antrum. We are not lessening the importance of the roentgenological examination by admitting these facts. It is important that we obtain the help of the clinician in all cases and also it is often necessary that the patient be gastroscoped. Even after all methods have been used it may be necessary to resort to an abdominal section before the diagnosis can be made. Carcinoma of the pyloric antrum may simulate benign lesions clinically and often show a considerable amount of improvement following careful medical ulcer regimen. Relief of symptoms may continue for some time, but during this time the malignant process usually continues to progress.

Camp[18] states that in the pyloric area carcinoma greatly outnumbers benign ulcer and that prepyloric ulcers are more prone to undergo malignant degeneration than ulcers elsewhere in the stomach. Holmes and Hampton state that they believe that

any chronic, indurated lesion occurring in the pyloric antrum within 1 inch of the pylorus, but not involving it, should be considered malignant until proved to be benign. They are of the opinion that it is advisable to surgically remove all such lesions rather than use palliative treatment. Doub,[30] reported 35 patients with lesions of the prepyloric and pyloric area of the stomach. All of the patients had had a resection of the pylorus and the antrum of the stomach. In every case the specimens were studied carefully by serial sections through the ulcerated area and a final microscopical diagnosis was available. The patients' ages varied from twenty-six to seventy-three, or an average of forty-six years. Some of the patients had compained of symptoms for over twenty years, with an average of eight years. Practically all of the patients had been subjected to medical management for varying periods of time.

Summarizing the 35 cases, the final diagnosis which was arrived at by microscopic sections: there were 24 cases of benign peptic ulcer and of these 16 were located in the prepyloric area, and 8 in the pyloric ring. There were 7 cases of carcinoma of the antrum, there were 3 cases of chronic gastritis, and 1 case of syphilis of the antrum. In the 7 cases of carcinoma the preoperative roentgen diagnosis was as follows: pyloric ulcer 2; prepyloric ulcer 2; narrowed antrum, but not characteristic, 3. The average age of these 7 cases was forty-seven years, and the age of the cases in the entire series was forty-six years.

Carcinoma of the stomach is the commonest tumor found in the body of the male, and it occurs in the female only slighly less frequently than carcinoma of the uterus. Carcinoma of the stomach may have an insidious onset and this increases the importance of early diagnosis. Any method that will lead to early recognition and proper surgical treatment offers the best chance of recovery. Radical surgical treatment is impossible and feasible only in the early stages of the disease and early recognition and operation offer the best chance for a cure. In the advanced stages of the disease its recognition is not difficult by the roentgenological examination. During the early stages of the disease it is much more difficult to recognize conclusively the nature of the lesion encountered, but even though the accurate diagnosis is not always possible by means of the roentgen ray, it probably offers the best chance at the present time of an early diagnosis.

Fig. 11. Patient C. W. Cancer of the pyloric antrum; deformity meniscus complex; proved at operation.

Gastroscopic examination by a skilled operator is possibly more accurate than the roentgenological examination. There are, however, blind areas to the gastroscope which may be missed even by a skilled operator. While the roentgen examination should not be competitive but complementary, it is advisable to have the roentgen examination precede gastroscopy. Certainly the esophagus and the fundus of the stomach should be carefully examined before gastroscopic examination is attempted, to rule out or exclude contraindications, such as far-advanced tumors, large diverticula and strictures. It has long been

stated by such authorities as C. H. Mayo, Wilson and MacCarty at the Mayo Clinic that many cancers supervene or have their origin on a simple peptic ulcer base. Moynihan, Sherren and others concur in and support this view. Schindler, however, holds a contrary view, and states that in a long series of simple gastric ulcers which he has gastroscoped for a number of years he has never seen one that has become malignant. For a number of years, MacCarty of the Mayo Clinic was of the opinion that 68 per cent of apparently simple ulcers showed microscopical evidence of cancer. Ewing, Dibble and others have placed a different interpretation on the histopathological appearance. Newcomb, using his criteria for the pre-existence of a simple peptic ulcer and an ulcerating carcinoma, found that only 13 per cent of the cancers showed evidence of an antecedent peptic ulcer, and that 3 to 7 per cent of the ulcers showed malignant changes. Clinical evidence would also tend to disprove the peptic ulcer cancer sequence. Whereas the average history of symptoms in the peptic ulcer is from five to seven years, in cancer it is usually in the neighborhood of six months. Another point against the possibility of the cancer having its origin in an ulcer is that the great majoity of the simple gastric ulcers are located on the lower curvature while the pyloric antrum is the commonest site for cancer. Balfour investigated the after-history of 1,280 cases operated upon for gastric ulcer. The operation of choice in those days was usually a gastroenterostomy and the ulcer was left in situ. He found, however, that less than six per cent of these cases died of cancer of the stomach. Stewart's analysis of 733 stomachs surgically incised for chronic gastric ulcer or cancer between 1921 and 1940 gave an incidence of 10 per cent, or 51 cases of cancer developed in a total of 510 chronic ulcers. It would seem from the above evidence that the incidence of degeneration of simple ulcer into a carcinoma is not as common as was formerly reported. Most authorities believe that not more than 10 to 15 per cent of gastric cancers arise from chronic ulcers. The infrequency with which carcinoma develops in duodenal ulcer, which is a much more chronic type than the gastric ulcer, would also seem to disprove the theory that an ulcer undergoes degeneration and becomes malignant.

Carcinoma of the stomach is usually described as scirrhous or medullary; encephaloid or fungus. The cells are usually spheroidal or columnar. The spheroidal type seems to be twice as common as the columnar, and is the frequent type of malignant ulcer. The columnar type is the commonest in the pyloric antrum. It is frequently of the fungoid type, producing a large growth. The entire stomach may be infiltrated by either type and produce what is commonly called the leather bottle stomach or linitis plastica. According to Sherren, the spheroidal cell variety often causes a marked diminution in the size of the stomach, while the columnar cell variety causes no diminution in the gastric size. Carcinoma of the stomach commences or has its origin in the mucosa and tends to spread to the submucous layer. Usually an indurated area marks the limits of the infiltration of the mucosa, whereas the submucosal infiltration may extend for several centimeters beyond. The spread of the disease may be due to the drainage glands of the stomach, directly to the liver or pancreas and the peritoneum generally by seeding, lastly by the blood stream to the liver, lungs, bones and brain. Metastasis of gastric carcinoma to bones is rather unusual as the patient usually dies before the metastasis occurs.

Roentgenographically carcinoma of the stomach may be classified as follows:

1. Scirrhous type, which may be localized or diffuse.
2. Encephaloid or fungus type.
3. Malignant ulcer.

The scirrhous type usually causes contracture of the stomach, causing a shallow or indistinct filling defect. The deformity may involve one or both curvatures, and the narrowing which it causes may be

minimal or maximal. The changes from the healthy to the infiltration in the stomach are often gradual, but may be abrupt. A napkin-ring deformity is the common type of filling defect encountered in the scirrhous type of carcinoma, constricting the gastric lumen. The closer the lesion is to the pylorus, the narrower the constriction, which usually causes obstruction. The configuration remains unchanged in a series of roentgenograms or during roentgenoscopic examination. There is a lack of peristaltic waves over the area of constriction. The waves pass downward and can be followed to the upper limit of the filling defect and then disappear. The portion of the stomach that is constricted shows an absence or a lessened flexibility. Usually the edges of the curvatures of the constricted area are smooth and present no jagged edges. Occasionally on palpation under the fluoroscope a mass may be felt. The mucosal pattern is usually altered in the presence of a scirrhous infiltration, but it is often difficult to expand the lesion enough to study the mucosal pattern. There are definite changes in the function of the pylorus in the presence of scirrhous carcinoma. If there is no mechanical obstruction the pylorus tends to be widely patent (gaping pylorus of cancer). This may be due to infiltration of the pyloric canal, transforming it into an open rigid tube. Usually infiltration will cause a stenosis with pyloric obstruction. If, however, there is an achylia the gaping pylorus is probably functional being associated with the achylia.

Medullary or encephaloid carcinoma may occur in any part of the stomach along the greater curvature of the pars cardiaca, pars media, or involve the entire pyloric antrum. The outstanding roentgen diagnostic feature is the presence of a large fungating filling defect. Accompanying the mucous and submucous infiltration is the formation of an irregular large tumor mass that extends into the gastric lumen. This type of tumor is prone to ulcerate. These large irregular masses cause gaps into the barium shadow due to projection of the tumor masses into the lumen of the stomach. They cause filling defects in the barium suspension not unlike finger prints. The edges of the curvatures are often jagged and spike like. There is an absence of peristalsis in the region of the tumor masses. Irregular filling defects often vary a good deal with the amount of barium mixture that is given to the patient. If the tumor is located in the pyloric antrum there is usually a rapid emptying and a gaping pylorus, and there is a marked lessening of the flexibility and mobility of the gastric wall. The presence of a palpable epigastric mass is more common in the encephaloid than in the scirrhous type of carcinoma.

Carcinomatous plaques may occur in the encephaloid type of carcinoma and they are often difficult to recognize. These plaques may occur along the greater curvature of the stomach or in the pyloric antrum. These are characterized by thickened and irregular mucosal folds that closely simulate a hypertrophic type of gastritis. Of differential diagnostic importance is the extent of the lesion. If it is widespread and involves a large part of the stomach it is more likely to be a simple lesion, such as hypertrophic gastritis. On the other hand, however, hypertrophic gastritis may be localized in one part of the stomach. Roentgen differentiation of these two lesions may be impractical and is usually impossible. It is unwise to make a serial roentgen examination at monthly intervals due to the fact that the lesions are often malignant and these patients should be subjected to gastroscopy and laparotomy.

A discussion of carcinoma of the pyloric antrum of the stomach would be incomplete at this time if I failed to mention the meniscus sign first described by Dr. Russell D. Carman and later expanded upon by Dr. B. R. Kirklin and at the present time referred to as the meniscus complex.

The meniscus complex occurs in the presence of an ulcerating carcinoma, on or near the lesser curvature. The lesion extends into the gastric lumen and when the

palpating hand passes over the lesion the built up tumor mass shows an area of decreased density and in the center of this area there is often a crater which represents the ulcerated area with increased density due to barium in the crater of the ulcer. There are no peristaltic waves passing over this area. Based on many examinations, followed by operation, the meniscus complex is found due to carcinoma extending into the lumen of the stomach and in the tumor mass itself is the ulcerated area. This accounts for the area that harbors the barium, whereas the tumor mass itself represents the built up edges where the barium is displaced. The meniscus complex is especially valuable in identifying small lesions. A few points which are important in the differential diagnosis of simple ulcer from a malignant ulcer are as follows:

Simple ulcer is usually on the lesser curvature and distant from the pylorus, whereas a malignant ulcer is variable in site, but most common on the lesser curvature in the pyloric antrum. Ulcers on the greater curvature are not common and are nearly always malignant. Therapeutic tests may be of value in differentiating the two types of ulcer. Simple ulcer will often heal in a few weeks under medical management. A malignant ulcer usually fails to heal. A malignant ulcer may for a time get smaller during treatment. This may be due to the lessening of the edema, resolution of the mucosa, contraction of the ulcer, or perhaps a filling of the crater by tumor tissue. In the presence of a simple ulcer the rugae tend to converge to the crater. In a malignant ulcer they are usually interrupted without conversion.

Sarcoma is said to account for 1 per cent of all gastric tumors. This tumor is very seldom diagnosed roentgenographically as it very closely resembles and simulates carcinoma, roentgenoscopically and roentgenographically. Sarcoma of the stomach usually occurs somewhat earlier than carcinoma, usually between twenty and thirty years of age, occurring very rarely after seventy. Very frequently there are no symptoms. Vomiting is probably the most common symptom and occurs in about half the cases. The presence of a palpable epigastric tumor is quite common and is met with more frequently than in carcinoma. Changes in acidity may occur but are less frequent than in carcinoma. Occasionally perforation and hemorrhage may take place. Pyloric stenosis with obstruction is rare and cachexia is a very late symptom. In a series of 58 cases the distribution of sarcoma was as follows: greater curvature 18; pylorus 14; lesser curvature 1; anterior wall 8; posterior wall 8; diffuse infiltration 4; and cardia 1. Histopathologically there are three types of sarcoma occurring in the stomach:

1. Round cell sarcoma, which has its origin in the submucous tissue and may be diffuse or localized. The tumor encroaches upon the gastric lumen. This type of tumor is most common in the pyloric half of the stomach and is often confused with an encephaloid carcinoma or a polypoid growth. This type of sarcoma represents about 60 per cent of the gastric sarcomas.

2. Spindle cell sarcoma is the next most common type, usually representing 36 per cent. This type of tumor is pedunculated and forms a subserous mass. Occasionally they become very large filling the entire abdomen and may be confused with ovarian tumor. There may be cystic degeneration accompanying this type of sarcoma.

3. Lymphosarcoma, or malignant lymphoma, tends to infiltrate the stomach widely and causes definite thickening of the wall of the stomach. Frequently other portions of the alimentary tract, such as the ileum and cecum, are involved simultaneously. Frequently the pyloric antrum is the site of the tumor. The pylorus itself is not usually implicated, and obstruction and stenosis are uncommon. Roentgenological findings are varied. The commonest finding is that of a filling defect with rigidity of the gastric wall and absence of peristalsis. There is nothing in these findings that serves as a distinguishing feature of diagnostic importance. In other words it is

impossible to differentiate a lymphosarcoma from a carcinoma roentgenographically.

Recently Gage has described what he considers to be a characteristic appearance in the submucous pedunculated lymphosarcoma of the stomach. He believes that these tumors tend to necrose at the center and produce a filling defect like a tangerine in the middle of which is a deep ulcer crater or niche. Lundquist has also reported a similar appearance in a neuroma of the stomach, a rare, usually benign tumor, poorly vascularized and tending to necrose. Most of these tumors are found on the greater or near the greater curvature.

THE BENIGN PYLORIC ULCER

A benign pyloric ulcer presents different roentgenographic features from those seen elsewhere in the stomach. A deep crater is seldom visible in the pylorus. This is due to the fact that the ulcer is enclosed in a muscular ring and no mucosal edema can develop. When seen in the pylorus, the crater is usually small and shallow; at times, it may be in the form of a spicule or tiny diverticulum. A rosette appearance may be seen from the central fleck and radiating plicae when the crater is seen en face. The pyloric canal is more commonly distorted; when the ulcer crater extends into the duodenum, as is commonly the case, there is deformity of the duodenal bulb. In many instances actual pylorospasm is present.

Indirect signs of the presence of a pyloric ulcer include pylorospasm, pyloric stenosis or obstruction, coarsened and irregular mucosal folds of the stomach and achalasia. Hurst described the last condition as a reflex inhibition of relaxation, where the initial opening of the pyloric canal is later than normal and the periodic relaxations are infrequent and relatively ineffective.

Therefore, the differential diagnosis of a benign peptic ulcer will embrace any condition which might cause pylorospasm, stenosis or obstruction. These include malignant lesion in the stomach, duodenal ulcer, cholecystitis, pancreatitis, renal or appendiceal lesion. Hypertrophic stenosis of the pylorus many occur and this poses another differential diagnostic problem.

According to Kirklin, an ulcer may be considered as probably benign when:

1. The symptoms have been present for ten years or more.
2. The patient is less than thirty years of age.
3. Free hydrochloric acid determination is 40 or more.
4. The lesion is located on the lesser curvature.
5. The ulceration extends through the gastric wall and is extraluminal.
6. The ulcer crater measures less than 2 centimeters.
7. The stomach is of the "B" type.
8. The biological test shows reduction of size of the crater under medical treatment.

SUMMARY

1. The pyloric antrum of the stomach extends from the incisura angularis to the pyloric canal.

2. Peristaltic waves are normally quite active in the pyloric antrum. Absence of peristalsis is a very important finding in the diagnosis of organic lesions.

3. The gastric mucosa is separated from the muscular layer by the tela submucosa and the mucosa moves independently of the muscularis.

4. The stomach receives its innervation from the vagi and the sympathetic system through the splanchnic nerves which follow the blood vessels. The two types of nerves form the myenteric or Auerbach's plexus, located between the longitudinal and circular muscular layers. This plexus controls peristalsis. The submucous or Meissner's plexus is located in the deeper part of the submucosa and controls the secretions in the stomach.

5. Most investigators believe that the pyloric antrum is the most common location for functional and organic lesions in the stomach.

6. Hypertrophy of the pyloric muscle cannot be classified as a common lesion and occurs about once in every 7,407 gastrointestinal examinations (Kirklin and Harris). Many authors believe that hypertrophy in adults represents a persistence of the infantile form. Hypertrophy is much more common in males than in females. Some authors report a sex ratio of 19 males to 1 female. The cause is still not definitely known. A number of authors believe that destruction of the ganglion cells may play a very important role as a causative factor. The condition is important in the adult as it may be very difficult to differentiate it from an infiltrative type of neoplasm.

7. *Antral gastritis* is thought to be a very common disease by many investigators, while others are of the opinion that it occurs infrequently. The cause of the condition is not definitely known but alcohol, tobacco and extremely hot fluids and food are said to be contributing factors. It is more common in males between the ages of forty and seventy. The symptomatology is variable and may simulate gastric ulcer, even to the point of hemorrhages. Wide, high and rigid rugae with hypersecretions are important diagnostic criteria. The roentgenologist and the gastroscopist are often in disagreement as to whether or not antral gastritis is present. The two methods should be used as complementary rather than as competitive methods and there should be close cooperation with the internist.

8. *Prolapse of the gastric mucosa* is not an uncommon condition and may exist when no other gastric pathology is present. The gastric mucosa frequently prolapses through the pylorus into the duodenal bulb with no other co-existing disease. The prolapse causes a deformity at the base of the bulb described as an umbrella-shaped mushroom. The folds are redundant and can be seen on the roentgenogram. Usually middle aged persons are affected and the symptoms are referred to the upper abdomen. Nausea and vomiting, and occasionally hemorrhage, are important manifestations. About 50 per cent of the patients are relieved on ulcer management. Surgery is seldom indicated.

9. Extragastric pathology causing deformities of the antrum is not uncommon and adds to the difficulties in differential diagnosis. The antrum is often smooth and elongated but may show a definite filling defect. Intragastric tumors, far advanced infiltrations and benign ulcerations are often more readily diagnosed than perigastric adhesions and bands. Extragastric narrowing and deformities may be due to congenital defects, falciform ligament, gastrohepatic ligament, and inflammatory processes, such as gallbladder pathology, postoperative adhesions and tumors of the pancreas and liver. The symptoms are often bizarre, often being similar to those of peptic ulcer while other patients may complain only of indefinite upper abdominal discomfort. Hemorrhage has been present in all of our cases (occult blood in the stools).

10. *Carcinoma.* About 75 per cent of carcinomas of the stomach are located in the pyloric antrum (Golden and Stout). Holmes and Hampton reported an incidence of 12 carcinomas to 1 ulcer in the prepyloric region. Prepyloric ulcers are more prone to become malignant than ulcers elsewhere in the stomach (Camp). Carcinoma of the stomach is the most common tumor found in the male and occurs in the female slightly less frequently than carcinoma of the uterus. In the light of our present knowledge, it would seem that the incidence of degeneration of a simple ulcer into a carcinoma is infrequent. "Less than 6 per cent of 1,280 patients with gastric ulcer after gastroenterostomy died of carcinoma. . . The ulcers were left in situ" (Balfour). Carcinoma of the stomach has its origin in the mucosa and spreads to the submucous layer. The disease may spread to the drainage glands of the stomach, direct to the liver or pancreas and the peritoneum by seeding or by the blood stream to the liver, lungs, bones and brain.

In closing, I cannot refrain from quoting Dr. Frank Lahey who in discussing prepyloric lesions said "I am certain that there

are pre-pyloric defects as demonstrated by roentgenograms that cannot be made to disappear by any known drug or plan of treatment. This being the situation, to find no lesion at exploratory operation when the roentgenologic defect is probably due to a pre-pyloric spasm is definitely better than at a later date to find that the lesion is malignant and that the favorable time for removal has passed. This is too high a price to pay for a simple exploration." This statement applies to any prepyloric lesion when the diagnosis is in doubt.

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LIPOID PNEUMONIA*

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PREVENTABLE diseases of most types have been eliminated or at least reduced by appropriate specific measures, but this is not true of lipoid pneumonia. Though this disease was first described in 1925,[7] no appreciable effort has been made to reduce its frequency. Many reports have appeared in the literature since its description, but a large number of oily preparations are still available on open purchase for self-medication. Physicians continue to prescribe oily nose drops for upper respiratory infections and mineral oil for constipation. Since these conditions still exist, there is need to review the clinical, pathologic, and roentgenographic findings in lipoid pneumonia.

Our interest in this disease began in 1948 with the futile attempt to treat a patient who was addicted to the use of nose drops. Prior to 1948 the diagnosis of lipoid pneumonia had been made only once at Garfield Memorial Hospital. Since that time 34 patients have been treated for the disease. The group is unique because 9 of the total of 35 patients were operated upon for lesions which simulated tumor,[2] and autopsies were obtained on all 10 patients who died of their disease.[4] In these patients the diagnosis of lipoid pneumonia was proved by the visualization of large amounts of oil in tissue sections. No other pathology was present in any of the lungs. The diagnosis in the 17 living patients was made on the basis of a history of oil ingestion, consistent roentgen findings, and the presence of intracellular oil in the sputum. It is possible that the patients in this group may have been misdiagnosed, but they have been followed for an average of two and a half years and none has shown any other pulmonary disease.

There was considerable doubt at the beginning of this study that the excised lesions found in surgical specimens and at autopsy were caused by ingested oil despite the absence of other etiologic agents. It was suggested that the oil present in the lungs was secondary to some degenerative process though no such process was found in any of the cases. In those patients who had a bronchogram the source of the oil was obvious, but large quantities of oil were present in the lungs of those who did not have bronchograms. Control fat stains were done on the lungs of 6 patients who had bronchograms prior to pneumonectomy for bronchiectasis or carcinoma. Oil was found consistently in the bronchioles of all the cases and in small quantities in the alveoli of some of the cases. The thin alveolar walls of the control group occasionally showed minute droplets of oil, but this was minimal compared to the oil in the alveoli and in the thick fibrous wall of lipoid pneumonia.

This series was too small to be of statistical significance, but it afforded an opportunity to correlate the roentgen and pathologic findings. The roentgenograms presented an extremely varied picture and when examined individually were usually not diagnostic. When examined together they could be roughly arranged in two categories: diffuse and nodular. The following case reports illustrate some of the more important aspects of lipoid pneumonia, one chosen from each group.

REPORT OF CASES

CASE 1. A thirty-five year old white female was admitted to Garfield Memorial Hospital in September, 1948, with a chief complaint of dyspnea. She had been well until eight months

* Former residents of this clinic who also contributed and gathered data are: Carl Harry Wallman, M.D., Edward B. McCabe, M.D., Alexander Goulard, M.D.

prior to admission when she began to lose weight and feel tired. Dyspnea and a chronic productive cough began four months prior to admission. The dyspnea progressed until the patient became bedridden two weeks prior to admission. She had used an excessive amount of oily nose drops for the past three years. Sputum examination showed a large amount of intracellular oil. Physical examination on admission revealed cyanosis, roughened breath sounds, moist basal rales and a temperature of 100.2° F. The fingers showed moderate pulmonary osteoarthropathy. A roentgenogram of the chest in July, 1948 (Fig. 1), showed diffuse, fine, linear densities throughout both lung fields. The admission film (Fig. 2) showed these densities to have become confluent, involving the entire periphery of the lung. The patient became progressively dyspneic and died on the seventh hospital day. Postmortem examination revealed the right lung to weigh 950 grams and the left lung 910 grams. The lungs were firm and rubbery and not collapsible. Histopathologically, most of the functioning lung tissue had been replaced by hyalinized fibrous tissue which contained innumerable small droplets of oil. Most of the remaining alveoli were reduced in size and contained conglomerate masses of oil, fat-filled phagocytes, and desquamated alveolar lining cells. In some of the peripheral alveoli the lining cells had become cuboidal, and the alveolar walls were thickened by diffusely arranged lymphocytes.

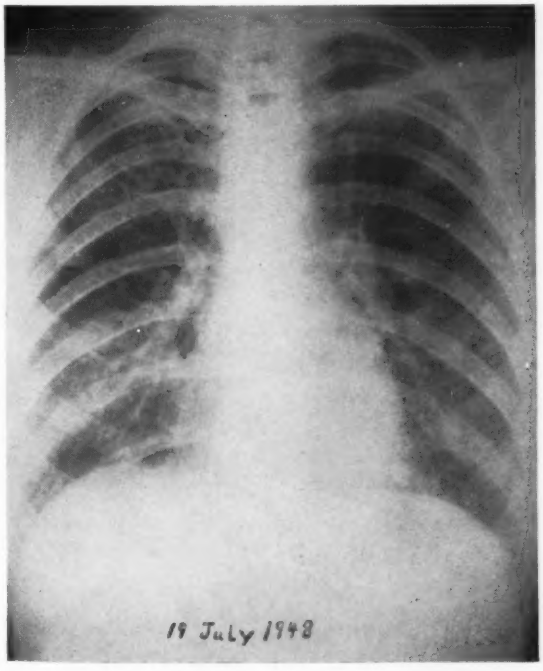

Fig. 1. Case 1. Roentgenogram shows diffuse, fine, linear densities throughout the lung fields simulating diffuse virus infection.

Fig. 2. Case 1. Two months later the densities have now become confluent leaving very little aerated lung and the patient is quite dyspneic.

Case II. A fifty-three year old white male was admitted to Garfield Memorial Hospital in August, 1950, complaining of a persistent cold. This had been accompanied by fever and increasing fatigue. During the past five years he had had a chronic, nonproductive cough, and he had been treated for pneumonia twice. Physical examination on admission was essentially negative except for dullness to percussion and fine moist rales in the right lower lobe bronchus. Cytology and cultures were negative.

Review of a roentgenogram of the chest taken in October, 1949 (Fig. 3), confirmed it as normal, but the roentgenogram taken on admission (Fig. 4) to the hospital demonstrated a nodular mass extending from the right 6th to the 12th rib posteriorly. Roentgenoscopic examination showed the mass to be fixed. A bronchogram showed normal filling of all the bronchi. The mass was thought to be either a lymphoma or an unusual appearing carcinoma.

At operation four days after admission the right lower lobe was uniformly firm. The entire

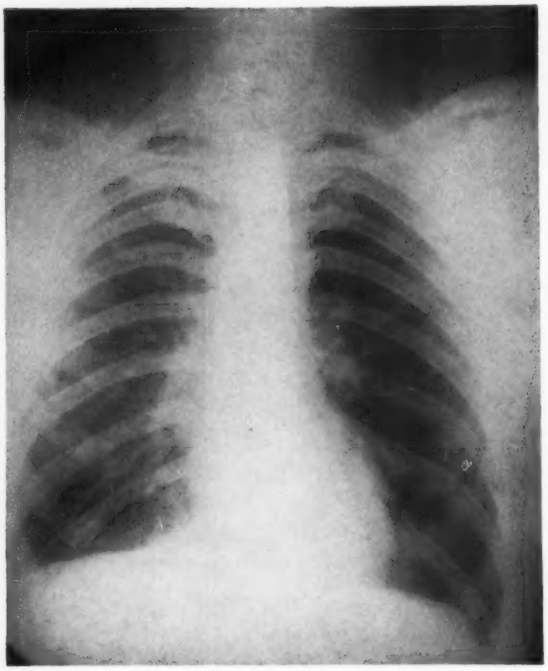

FIG. 3. Case II. Normal chest.

lung was densely adherent to the chest and diaphragm, and the hilar nodes were greatly enlarged. A pneumonectomy was accomplished with difficulty because of many adhesions. The lung weighed 1,000 grams. The entire surface was covered by fibrous tags. It was partially collapsed and bluish-red in the upper lobe, but it was solid and grayish-brown over the lower lobe and inferior portion of the middle lobe. The interlobar fissures were indistinguishable. Cut section showed uniform, spongy, yellowish-gray tissue throughout the entire lower lobe and approximately half the middle lobe. The bronchi were slightly reduced in diameter but were patent throughout. Microscopic sections of the lower and middle lobes showed alveoli devoid of lining cells and surrounded by wide bands of fibrous tissue. The alveoli contained large masses of desquamated alveolar lining cells, debris and lipophages. Sections stained with Sudan III showed small particles of sudanophilic material in the fibrous stroma and large quantities in the alveoli.

The patient died following the operation. Autopsy showed small amounts of oil in the left lung and some of the alveolar lining cells were cuboidal, but there was no increase in fibrous tissue in the alveolar walls. After the death of the patient, it was discovered that he had taken mineral oil as a laxative for ten years. It was ascertained that he took a large amount of mineral oil before retiring every night and also upon arising in the morning. It was also

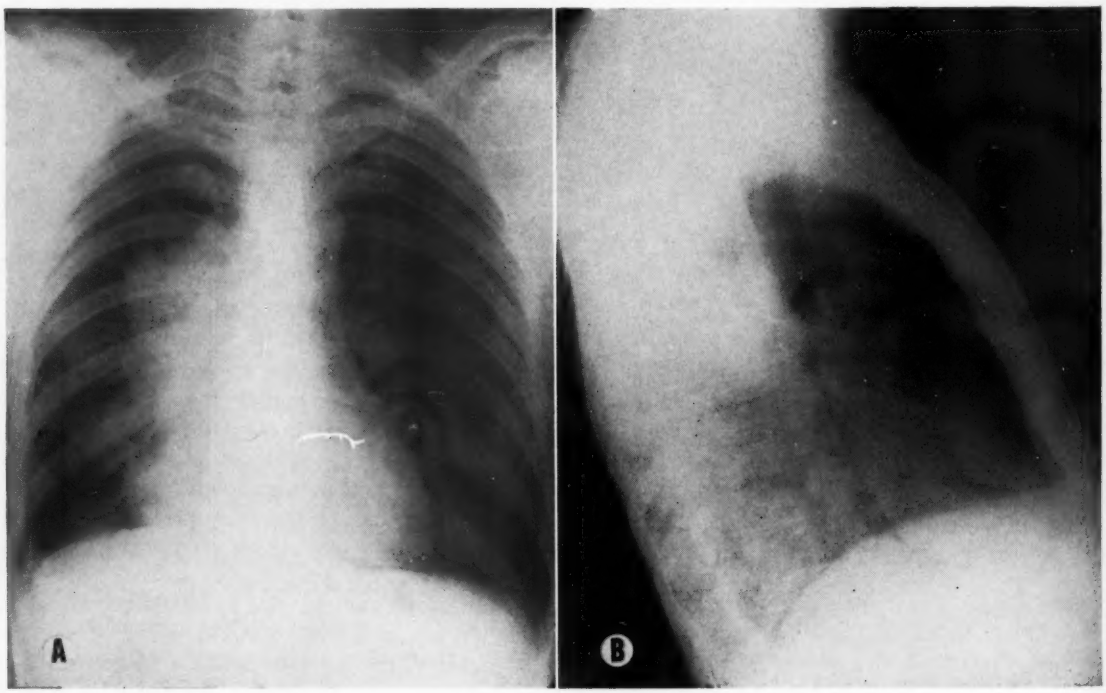

FIG. 4. Case II. (*A*) and (*B*) Ten months later a large nodular mass has appeared in the posterior aspect of the right lower lobe simulating tumor.

learned that he always went to sleep lying on his right side.

ETIOLOGY

Lipoid penumonia is caused by the aspiration of animal, vegetable, or mineral oils.[8] The oils which produce most of the cases of lipoid pneumonia in adults are bland and are usually combined with some agent considered to have medicinal value. Following either oral or nasal administration of oily material, portions of it remain adherent to the pharyngeal mucosa. Part of this drains into the esophagus, but the remainder sometimes drains into the trachea. If this occurs repeatedly, enough oil collects in the alveoli to produce pathologic changes since no method is known for the removal of significant amounts of intra-alveolar oil. It has never been determined how certain individuals can ingest large amounts of oil over long periods without developing lipoid pneumonia while others die of the disease after having used relatively small amounts. An attempt was recently made to explain this difference. Four volunteers in the Department of Radiology tried to aspirate oil into their lungs by sipping lipiodol while in the supine position. All four volunteers experienced considerable choking and coughing followed by nausea, but roentgenograms of their chests failed to show any oil in the trachea or bronchi. It has been assumed that the occurrence of lipoid pneumonia depends to some extent on individual susceptibility. This theory is very difficult to prove and it may not be true. Unlike the four volunteers, some individuals do not have an active gag or cough reflex. The presence of an adequate cough or gag reflex may be the factor preventing lipoid pneumonia even though large amounts of oil are ingested.

The prevalence of lipoid pneumonia is not known because no standard criteria have been developed for establishing the diagnosis, and opinions regarding criteria are widely divergent. The frequency of occurrence depends to a great extent upon the diligence with which it is sought. In Table 1 is listed the prevalence ratio recorded by five investigators.

Table 1

POSTMORTEM PREVALENCE OF LIPOID PNEUMONIA

Reported by	Year	Consecutive Postmortems	Cases of Lipoid Pneumonia	Percentage
Laughlen[7]	1925	300	1	0.3
Pinkerton[10]	1927	290	6	2.0
Ikeda[5]	1937	101	7	6.9
Bromer and Wolman[1]	1939	137	12	8.7
Freiman *et al.*[3]	1940	3,500	41	1.4

Though the data in this table appear to be comparable, lipoid pneumonia was only an incidental finding in many of the patients and not the cause of death. At Garfield Memorial Hospital lipoid pneumonia was the cause of death in approximately 1 per cent of the last thousand autopsies. In other large series of autopsies lipoid pneumonia is not listed as a cause of death nor as an incidental finding in a single case. Terms such as "idiopathic pulmonary fibrosis," "diffuse interstitial fibrosing pneumonitis," or "Hammon-Rich syndrome"[6,9,11] have occasionally been listed. Many of the published photomicrographs illustrating the pathology of these conditions are very similar to those of lipoid pneumonia. It has not yet been found necessary to use these terms at Garfield Memorial Hospital.

In children the disease is usually produced by animal oil in the form of fish oils administered for their vitamin content or by butter fat in regurgitated milk. Children who vomit their feedings and those with defective deglutition are most susceptible to the disease.

Most of the cases in this series were adults and, as in larger series, the etiologic agent responsible for the disease was mineral oil taken to combat constipation. Lipoid pneumonia was produced by the voluntary ingestion of mineral oil in 22 patients, by nose drops in 8 patients, by milk and cod liver oil in 2 infants and by petroleum jelly in 1 instance. No history of oil ingestion was obtainable in 2 patients. Such a history is not always easily obtained.

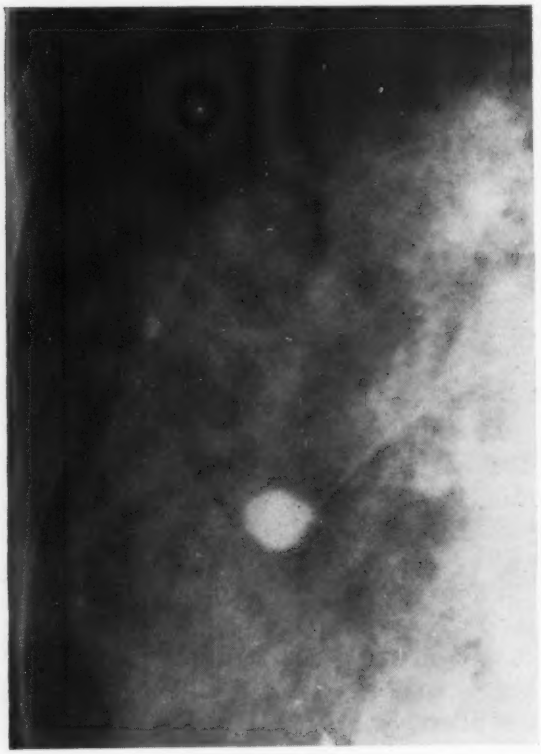

FIG. 5. Magnified localized view showing the diffuse, fine, linear, spun-glass densities.

Patients will usually not admit to using nose drops or spray containing cocaine. Others may not realize that the medication they are using contains cocaine. Cocaine was combined with oily nose drops in the case of 2 physicians and in 3 others.

CLINICAL FINDINGS

Only 24 patients in this series sought medical advice because of their symptoms. The others were referred for investigation because of asymptomatic lung lesions found on routine examination. Most of the patients had vague indefinite complaints and no single complaint was common to all patients. Cough, dyspnea, and chest pain were the most frequent complaints. One adult had recurrent hemoptysis and both children had recurrent attacks of pneumonia. As in larger series there were almost an equal number of males and females, there being 19 women and 16 men. The ages of the patients ranged from three months to eighty-six years, the average being fifty-two years. Only 2 of the 35 patients were Negroes. It is known that lipoid pneumonia is a frequent finding in autopsies on debilitated patients. However, none of the patients in this series was debilitated and, prior to the present illness, all were well and without complaints.

The physical signs in lipoid pneumonia were not specific and varied with the size, position, and character of the lesion. The most common physical findings were dullness to percussion, harsh breath sounds, and bronchovesicular or bronchial breathing. One patient showed pulmonary osteoarthropathy. A low grade fever was present in several of the patients with diffuse disease. Clinical laboratory tests were unrewarding and served only to rule out other diseases.

ROENTGEN FINDINGS

A definite diagnosis of lipoid pneumonia could not be established from the roentgen appearance alone. Although the densities produced by this disease varied in their size, shape and location, they could be grouped into two definite types: diffuse and nodular. Either type may lead to collapse of a lobe, necrosis, or bullae formation and pneumothorax. Abscess with fluid levels was not demonstrated and the term central necrosis was thought to be more appropriate.

The diffuse group, comprising 24 cases, was characterized by diffuse, fine, linear, spun-glass densities, the periphery of which sometimes was fairly sharp but more commonly faded out into the lung fields (Fig. 1, 2 and 5). These densities were clustered together and intermingled with them were areas of rarefaction representing aerated lung. The general impression gained from these densities was that of fibrosis (Fig. 6).

There were 11 cases in the nodular group. The nodules were characterized by rather homogeneous, soft tissue densities varying from several centimeters in diameter to very large masses. The borders in some instances were sharply defined but in others they were irregular with extension of fine projections into the adjacent tissues. The

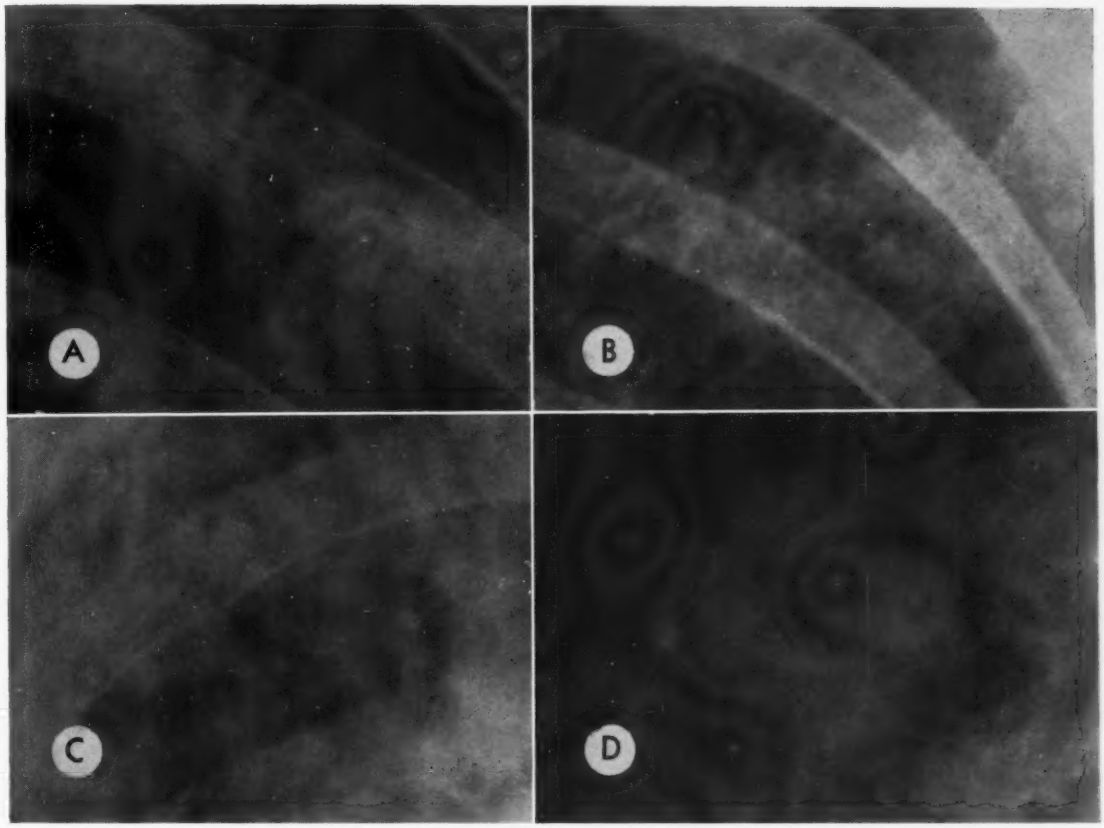

Fig. 6. (*A*) Silicosis. (*B*) Tuberculosis. (*C*) Idiopathic fibrosis. (*D*) Lipoid pneumonia. Note in *A*, *B*, and *C* the fibrotic densities are coarse, whereas in *D* the linear densities have a fine, spun-glass appearance.

large lobulated masses (Fig. 4) were apparently due to coalescence of multiple smaller areas.

When a fine focus tube and Bucky grid were used, these areas showed a spun-glass appearance and this proved helpful in differentiating lipoid pneumonia from tumor or other lesions. Roentgenologically, the lesions sometimes decreased in size and became denser due to fibrosis and shrinkage of the lung, and the patients appeared clinically improved (Fig. 7).

Five of the cases showed occlusion of the major bronchus to a lobe, usually the right middle lobe (Fig. 8*A*), 3 in the nodular group and 2 in the diffuse category. A one month old infant showed collapse of the right upper lobe following the aspiration of milk. The middle lobe was completely collapsed in 4 instances. The middle lobe bronchi could not be identified in one of the surgically excised lungs because of large areas of necrosis.

Bronchography was performed on 12 cases and the most common finding was obstruction of the terminal bronchi in the involved area. The main bronchus to the affected lobe did not always fill with oil, if the lobe was collapsed or when a large nodular mass was present distally in the parenchyma (Fig. 8*B*). This occlusion was due to granulation tissue in the bronchus in 1 case and in another case the bronchus was occluded by a broncholith, while distal to it was a large nodular mass of lipoid material. The cause of the obstruction was not established in 3 cases. In only 1 instance was the presence of bullae found, although this has been reported as occurring and leading to pneumothorax. In none of these cases was pneumothorax a complication. Pleural effusion associated with this

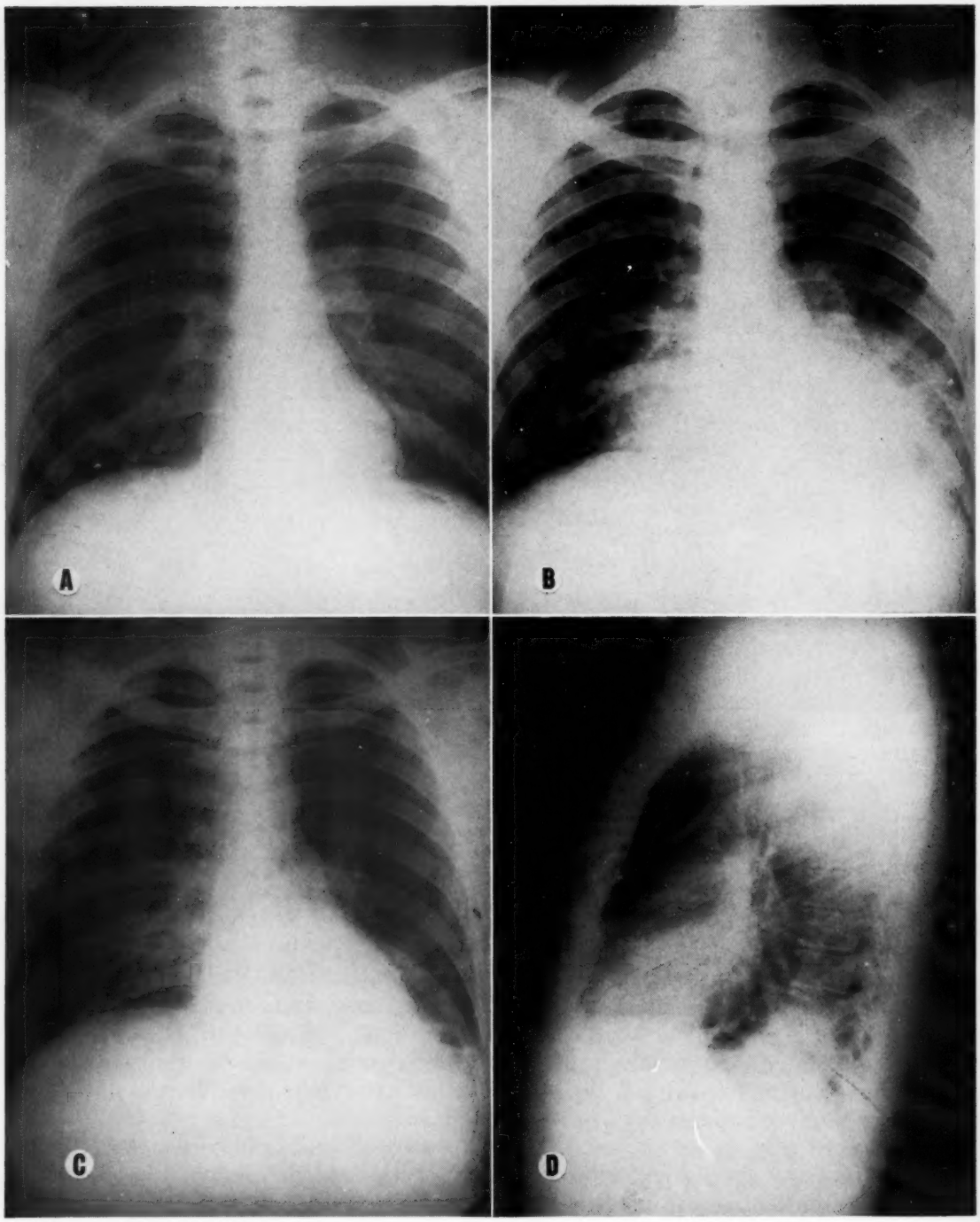

FIG. 7. A thirty-two year old physician who used oily nasal spray intermittently for eight months at the recommendation of an otolaryngologist. (*A*) 1946. Normal chest. (*B*) 1948. Large confluent densities have appeared in both lower lung fields. (*C*) and (*D*) 1954. The densities in the lung have become smaller due to fibrosis and shrinkage of the lung. The middle lobe is unusually small and thin. The patient is now symptom free.

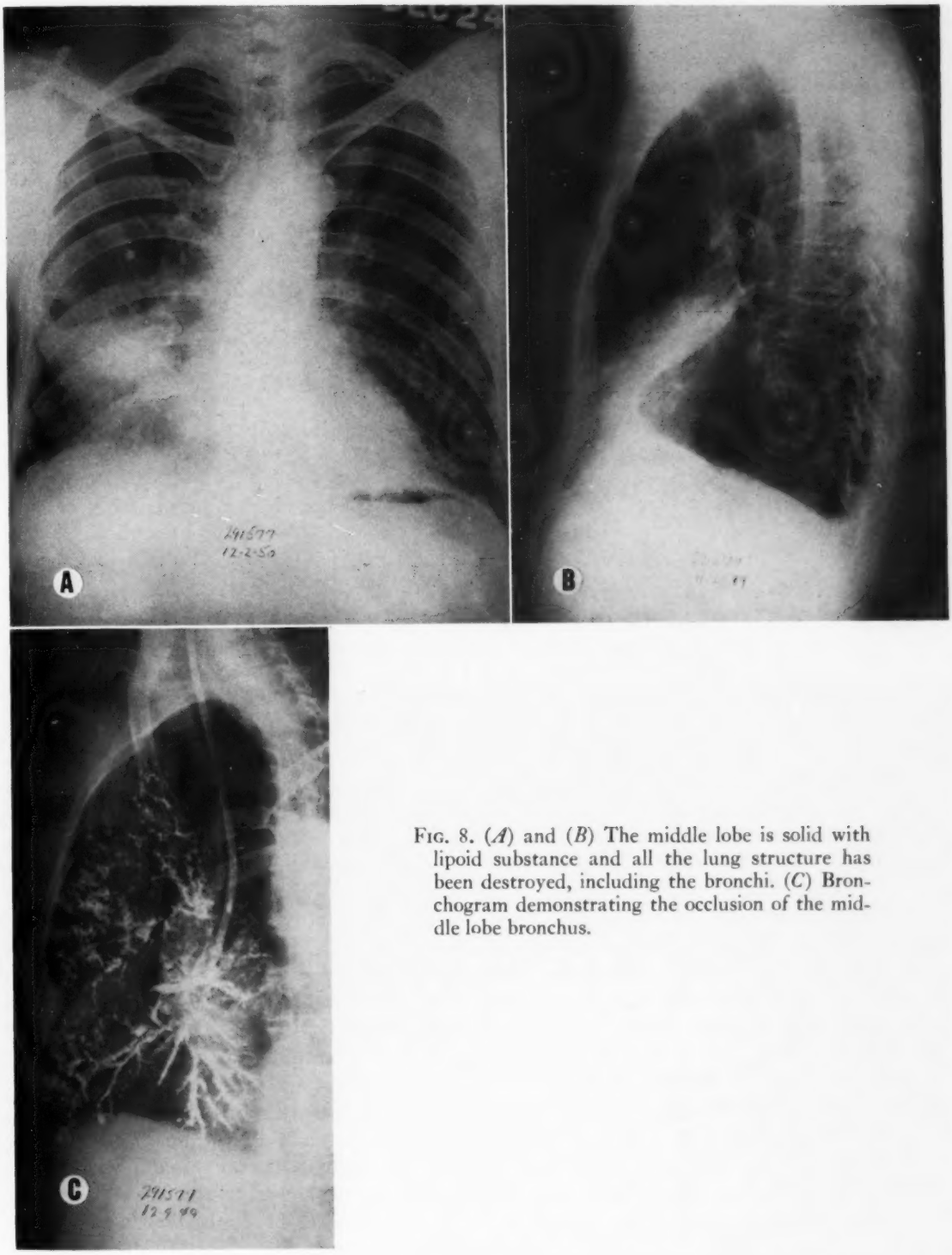

Fig. 8. (*A*) and (*B*) The middle lobe is solid with lipoid substance and all the lung structure has been destroyed, including the bronchi. (*C*) Bronchogram demonstrating the occlusion of the middle lobe bronchus.

disease was very unusual and only 2 cases showed small collections of fluid in the costophrenic angles.

Some of the investigators have been hesitant to make a diagnosis of lipoid pneumonia unless the pulmonary shadows were

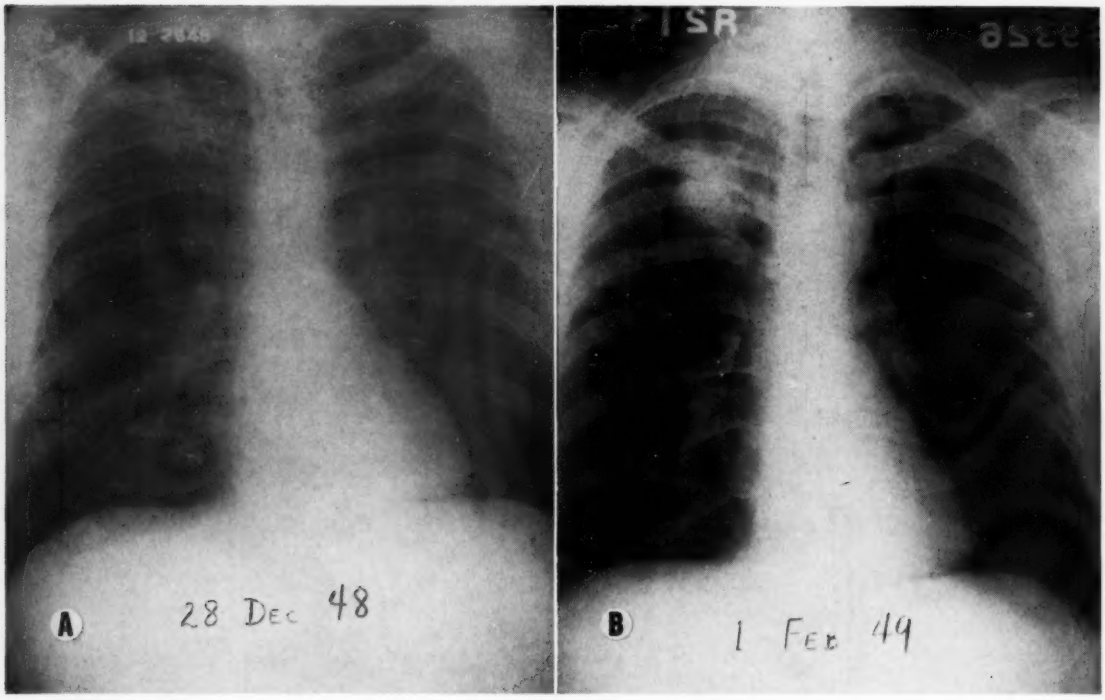

FIG. 9. (*A*) Area of increased density in the right upper lobe. (*B*) Thirty-four days later the density has become larger, airless and more discrete, simulating a tumor.

bilateral and fairly symmetrical. In this series both lungs were involved in only 21 cases. In the 14 cases with unilateral involvement, the disease was on the right in 12 cases, and on the left in 2 cases. The nodular areas were usually single (Fig. 9), but multiple areas were not uncommon and different lobes were involved (Fig. 10). The lower lobes were most often involved apparently because of the anatomical course of the bronchi but the axillary portion of the upper lobe was also frequently affected. It was assumed that this unusual distribution of oil, especially in the right upper lobe, could be due to ingestion of oil immediately before retiring. In infants developing the disease as the result of aspiration of milk or cod liver oil, the position in which the child was held during feeding possibly determined the location of the disease.

It has been assumed that after the disease became well established no regression was possible. In most cases this was probably true. No satisfactory follow-up studies have been done to confirm this. The present series followed for an average of only two and one-half years is certainly inadequate; but 3 of these patients have stopped taking oil and have shown some clinical and roentgenological improvement. Roentgenologically, the improvement sometimes appeared more marked because of shrinkage of the involved portion of the lung followed by compensatory emphysema. There was usually progression of the disease if the practice of taking mineral oil or oily nose drops was continued. After the right lung had been removed in 1 patient because lipoid pneumonia simulated a tumor (Fig. 11*A*), the patient persisted in using mineral oil, and seven years later the left lung showed similar involvement (Fig. 11*B*).

Roentgenologically, the most common error was that of confusing lipoid pneumonia with tumor. Lymphatic spread of carcinoma and pulmonary edema were other common conditions which were frequently confused with the disease. It was found that lipoid pneumonia was frequent-

ly confused with tuberculosis, atypical pneumonia, lobar pneumonia, infarction, Boeck's sarcoid and bronchiectasis.

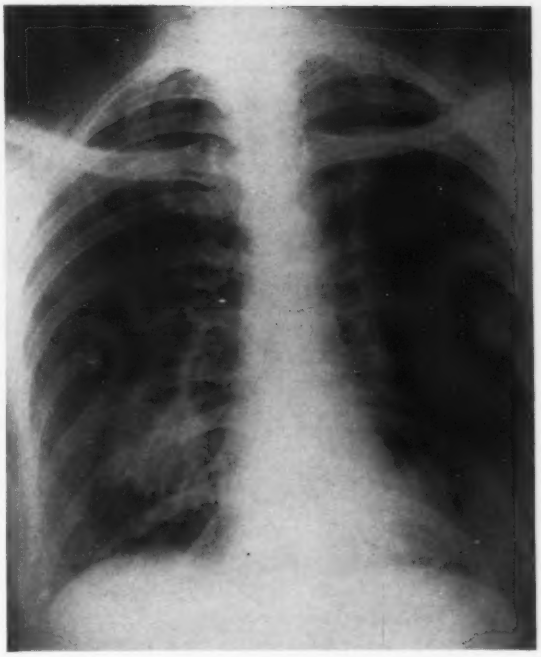

Fig. 10. Roentgenogram showing multiple localized densities in each lung.

PATHOLOGY

The gross and microscopic findings were based on the examination of 19 patients whose lungs were available following surgical removal or at autopsy. The variations encountered were dependent on the type of oil ingested, and the size, shape and position of the lesion. For purposes of description the cases were grouped into diffuse and nodular types.

In the lungs with localized lesions which appeared nodular roentgenographically, the palpable mass was often indistinguishable from carcinoma. The surfaces of these lungs showed no abnormality except for dimpling in the few cases where peripheral tissue was involved. The lungs diffusely involved were firm and subcrepitant, and

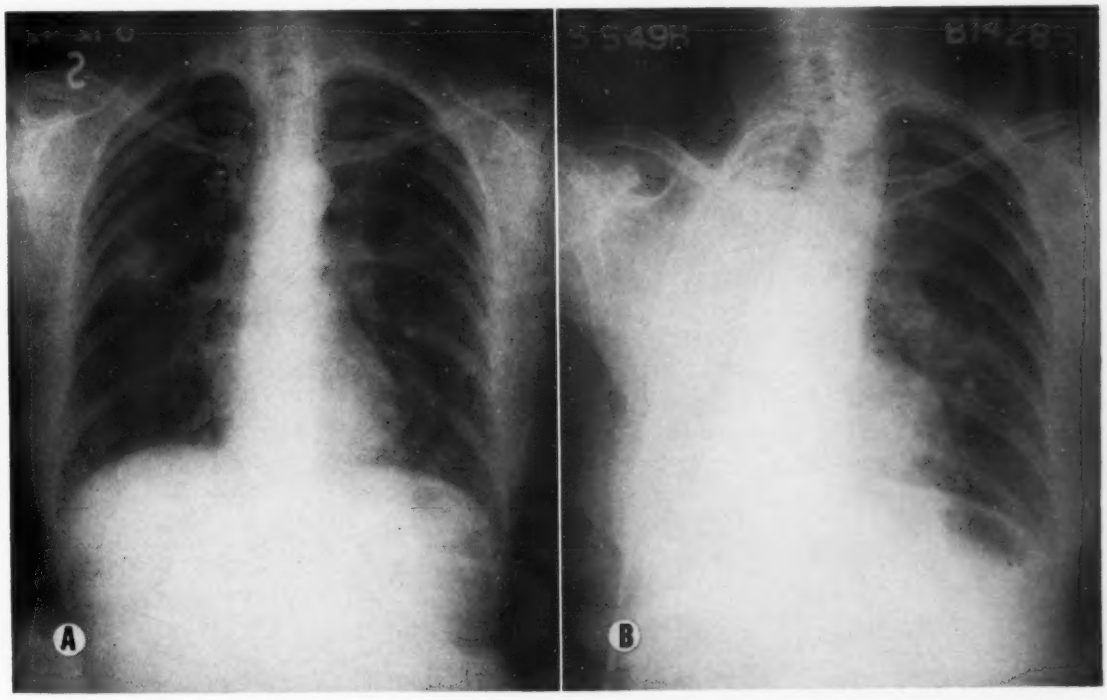

Fig. 11. (*A*) Roentgenogram demonstrates a solitary nodular mass in the right lung. The right lung was subsequently removed because the lesion was thought to be a tumor. The left lung is clear. (*B*) Four years later lipoid pneumonia has appeared in the left lung because the patient continued to use mineral oil.

FIG. 12. Early lipoid pneumonia. The alveoli are filled with lipophages and the alveolar lining cells are cuboidal.

their surfaces were mottled yellowish-gray and black.

Cut sections were characteristically firm, rubbery and yellowish-gray in the involved areas. The diseased tissue was well delineated and easily recognized in the nodular cases, but was ill defined and less prominent in the diffuse group. After fixation in formalin the distinction between diseased and normal lung tissue became more pronounced. The lungs of the 2 children and 2 adults showed areas of necrosis. These areas were small in 4 instances, but in 1 adult nearly half the lung tissue was necrotic and special stains showed that two types of oil were present. Animal oil of an unknown type was present in the necrotic areas, and the fibrous areas contained mineral oil.

Histologic sections did not adequately explain the presence of the fine linear densities seen so universally on the roentgenograms of patients with lipoid pneumonia. Peribronchial lymphocytic infiltration and beginning fibrosis which might have accounted for such an appearance were seen only in the areas of early involvement. The nodular and diffuse types of lipoid pneumonia were quite similar histopathologically. In the adults of this series the microscopic appearance varied with the degree of involvement.

In the early phase of the disease most of the alveolar lining cells were cuboidal and the alveoli contained partially phagocytosed oil (Fig. 12). Blood vessels were prominent and the intra-alveolar capillaries were thin walled and dilated. Fat stains on these sections showed sudanophilic material in the alveoli and also within phagocytes in the alveolar walls. A lymphocytic infiltration appeared later which greatly thickened the alveolar septa and reduced the size of the alveoli.

In the late stages (Fig. 13), large segments of functioning lung were replaced by fibrous tissue containing aggregates of lymphocytes, small oil droplets and scattered plasma cells. Most of the alveoli had disappeared. The remaining alveoli were represented by rounded spaces which were lined by fibrous tissue and contained conglomerate masses of desquamated alveolar lining cells, oil and debris. Vascularity was minimal. Small blood vessels were scattered throughout the fibrous stroma but showed no close relationship to the alveoli. Special stains showed loss of elastic tissue in all but the large vessels.

The only pathologic finding in living patients was the intracellular oil in the sputum. Oil introduced into the alveoli was quickly emulsified and phagocytosed. For this reason and because of frequent sudanophilic contaminants in submitted sputum, the pathologic diagnosis of lipoid pneumonia was made only when oil was found within the phagocytes. Considerable care was taken in the collection of sputum from suspected patients for the detection of oil. It was found best to collect the sputum be-

fore breakfast. This eliminated the difficulty in distinguishing histopathologically between phagocytosed oil and extraneous sudanophilic material.

The patients were instructed to collect only the sputum coughed up from the lungs. The specimens were collected in a clean glass container and were stained and examined promptly before degeneration of the fat-filled phagocytes occurred.

TREATMENT

No therapeutic measure now known is of help in evacuating oil from the lungs. After the fibrotic changes have occurred, no alteration to the normal is possible. Most patients who use oily medications apparently have no need for active treatment. Patients who develop symptoms should be advised to stop medication. Three patients in this series improved clinically after discontinuance of oily medication. The best treatment is prevention.

SUMMARY

1. The clinical, pathological and roentgenographic findings are reviewed in thirty-five cases of lipoid pneumonia.

2. The etiologic agents causing this disease include: mineral, animal and vegetable oil.

3. The cases are classified roentgenographically into diffuse and nodular types.

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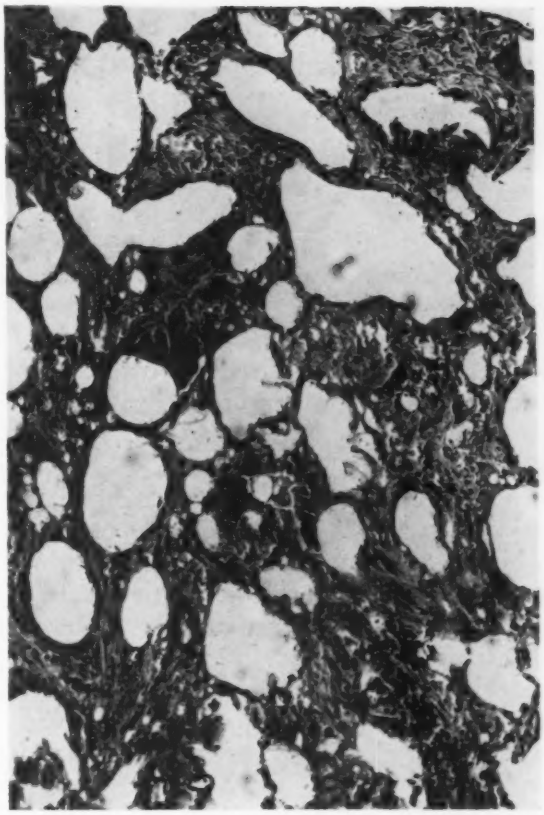

Fig. 13. Late stage of lipoid pneumonia. The alveolar walls are thickened and the alveoli are lined by fibrous tissue.

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DEXTROCARDIA AND UNSUSPECTED ABSENCE OF THE RIGHT PULMONARY ARTERY IN AN ADULT DEMONSTRATED BY ANGIOCARDIOGRAPHY

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PÔRTO ALEGRE, BRAZIL, S. A.

THIS unusual case demonstrates once more the rewards of a thorough examination, the tremendous potential of surprises in medicine and the utmost importance of careful appraisal of any abnormal findings which do not fit recognized morbid patterns. Although the case to be reported below is in no way a common one, we can accept it as a reportable instance which helps to prove that we must always make as complete an investigation as possible before establishing a definite diagnosis. Those who doubt this old and wise medical teaching do not proceed with their investigations far enough to challenge their simplest and first diagnostic impressions and so have insufficient opportunities to change their minds.

REPORT OF CASE

J.B.F., male, aged thirty-four, a sergeant of the Brazilian Air Force, consulted Dr. F. Dias Campos during a period of intense, transfixed pain localized in the lower third of the sternum which radiated to his shoulders obliging him to avoid any movement. This same pain had afflicted him twice in the last twelve years; each time it lasted half an hour, it appeared suddenly, it displayed each time the same characteristics and vanished independent of any recognizable therapeutic measure.

The patient's past history was not contributory. A complete physical examination did not show any important findings except some questionable reduction in the amplitude of respiratory movements, in the breath sounds and tactile vibrations in the right hemithorax. He admitted having had some shortness of breath since his early years which was never a definite handicap. Electrocardiograms were taken during and after the disappearance of the pain and were of no value (Fig. 5). Besides what is seen in the standard frontal and lateral roentgenograms (Fig. 1 and 2), a roentgenoscopic examination showed a respiratory pendulum: during expiration the right hemithorax experienced a further reduction and the right mediastinal shift increased; during full inspiration the right hemithorax increased and the mediastinum moved to the left, reaching almost the midline. These dynamic observations are shown in the respiratory roentgen kymogram (Fig. 3). There are no abnormal cardiovascular movements (teleroentgen kymogram, Fig. 4). Nowhere can we see a clear cut picture of emphysema; the aspects of the left vascular markings preclude absolutely the admission of obstructive emphysema in the left side. There are no abnormal masses which could be held responsible for the mediastinal displacement. The bifurcation angle is disputably broader than normal and this finding has misled those who have seen these films.

All these findings could not be explained rightly by the accepted opinions concerning mediastinal shifts and respiratory pendulum. Recognizing the definite and so far unexplainable reduction in volume of the right lung, we proceeded with the investigation of the bronchial tree. Laminagrams were taken with the patient erect. These laminagrams (Fig. 6 and 7) were taken respectively at 8 and 9 cm. from the posterior plane; the former in full inspiration and the latter in full expiration in order to exaggerate the right mediastinal shift and to show better what seemed to be a reduction in the caliber of the right superior lobe bronchus. This reduction can be appraised chiefly from the sketches but are very clear in the originals.

The bronchograms (Fig. 8, 9 and 10) definitely show a pliable, concentric, sometimes irregular, reduction in the diameter of the right superior lobe bronchus which is fully demonstrated in the bronchogram (Fig. 10) which was taken in full expiration. A direct bronchoscopy was done some days later and the bronchoscopist reported normal findings. At our request he made a bronchial washing in which neoplastic cells were found by the pathologist.

We were then seriously considering a thoracotomy when Dr. Paul H. Holinger happened to be in our city and we were able to show him this case. At our request, he kindly did a very minute bronchoscopy inclusive with his telescope and all who observed this examination could see that the right superior lobe bronchus appeared practically normal. Dr. Holinger told us that all was normal but the diameter of the right bronchial tree was everywhere less than the average. A new bronchial washing was done and in this liquid the same neoplastic cells were again found by the same pathologist.

Before recommending a surgical investigation we made angiocardiograms. It was a great surprise to discover the absence of the right pulmonary artery (Fig. 11 and 12). In the angiocardiograms (we obtained 6 angiocardiograms timed according to ether and decholin circulation times, which were normal) there is no trace of contrast medium in the right lung vessels even when, at the left, the vessels are shown in full detail with contrast medium even in the smallest branches.

With such a discovery, which explained completely the strange roentgen findings, we gave up our first idea of an operation and so we accepted the fact that the finding of neoplastic cells in the bronchial washings could be considered a pitfall. A thorough roentgenographic investigation of the cervical and thoracic spine did not explain the patient's pain. The narrowing seen in the bronchograms in the right superior lobe bronchus could be explained by a cartilaginous malformation of the bronchial wall.

We recommended to the patient that he watch for any future occurrence which could be interpreted as a progress of a lung neoplasm and suggested to him that he have frequent roentgen examinations. This he has done. We have noted no changes in the roentgenograms of his lungs. More than two years have passed since then. The patient's health is fairly good, and he has had no more pain. We thought it would be of interest to publish this remarkable case.

Comment. It is undeniable that this represents an absence of the right pulmonary artery. The right lung must receive all its vessels from bronchial arteries which were

Fig. 1. Posteroanterior view. Mediastinal displacement to right (dextrocardia in a broad sense or dextroversion). Retraction of the intercostal spaces and unusual elevation of the diaphragm leaf on the right side. Note the increased size of the left hilus and normal translucency on both sides. The right hilus is not seen, which is a common finding in cases of mediastinal displacements. There are no abnormal masses.

Fig. 2. Same case as Figure 1. Lateral view. The size of the pulmonary artery shadow is unusually large.

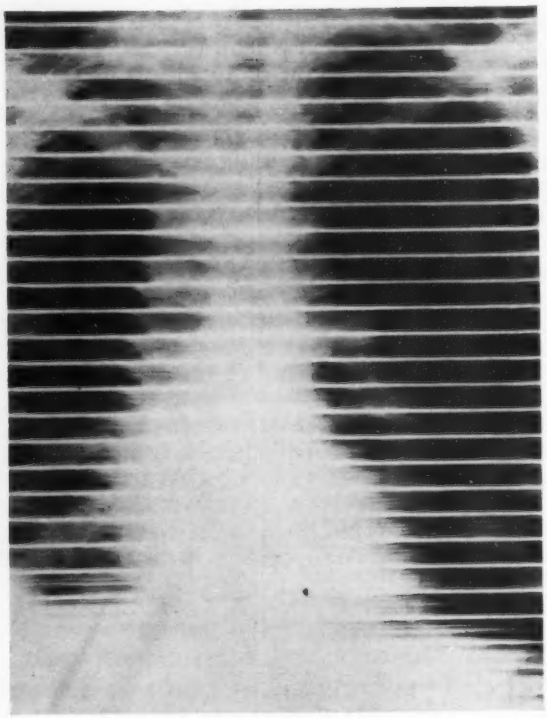

Fig. 3. Respiratory roentgen kymogram in posteroanterior position. For explanation see text.

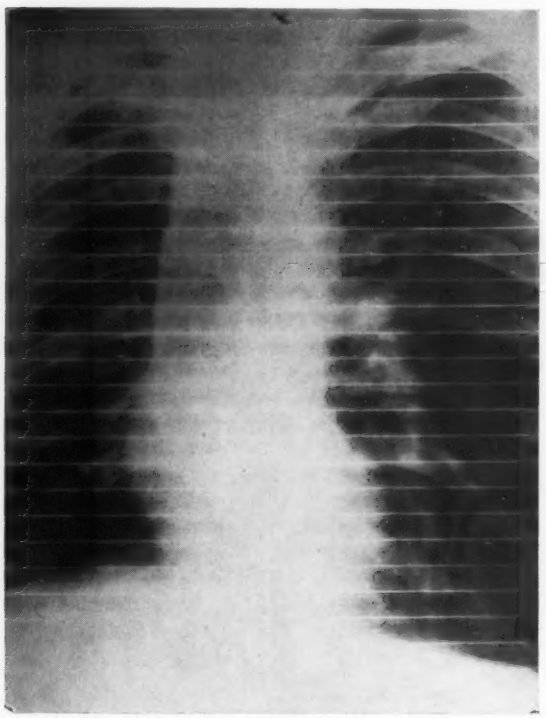

Fig. 4. Teleroentgen kymogram, posteroanterior view. There are no abnormal movements.

not shown in the angiocardiograms as they filled together with the aorta and must be of small diameter. This lung is functionless and hypoplastic. The left one has attained an enormous size and pushed the mediastinum to the right; as a matter of fact, the left lung invades the whole breadth of the mediastinal shadow, as can be seen following the contrasted left arteries in the angiocardiograms. Even in the reproduction of the roentgenograms without contrast material we can see that there is no right hilus. Before angiocardiography it was thought that the right hilus was hidden by the displaced cardiac shadow. Since no other reason could be found to explain the absence of the right pulmonary artery we think it can be admitted as a defect in the sixth aortic arch development.

We could find no mention of a similar case in the available literature. Recently, we found mention of a paper[4] under the title, "Abnormal arterial blood supply of right lung (right pulmonary artery originating from aorta) with normal blood supply of left lung from pulmonary artery," and published in a German journal in June, 1949. Unfortunately this paper is not available. Its title suggests, at least, that the general pattern of the pulmonary circulation in that article is similar to the one de-

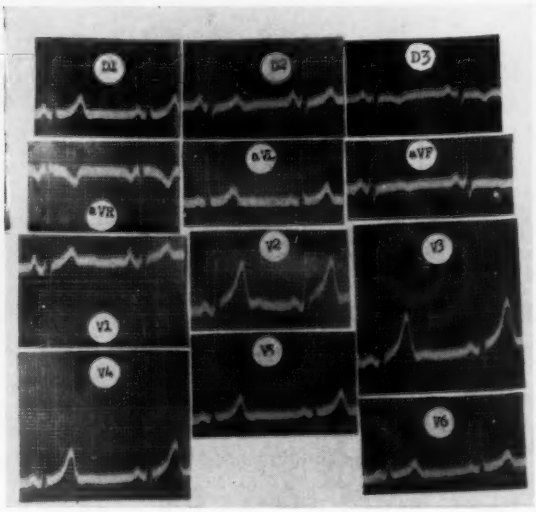

Fig. 5. Electrocardiogram. There are no definite abnormalities.

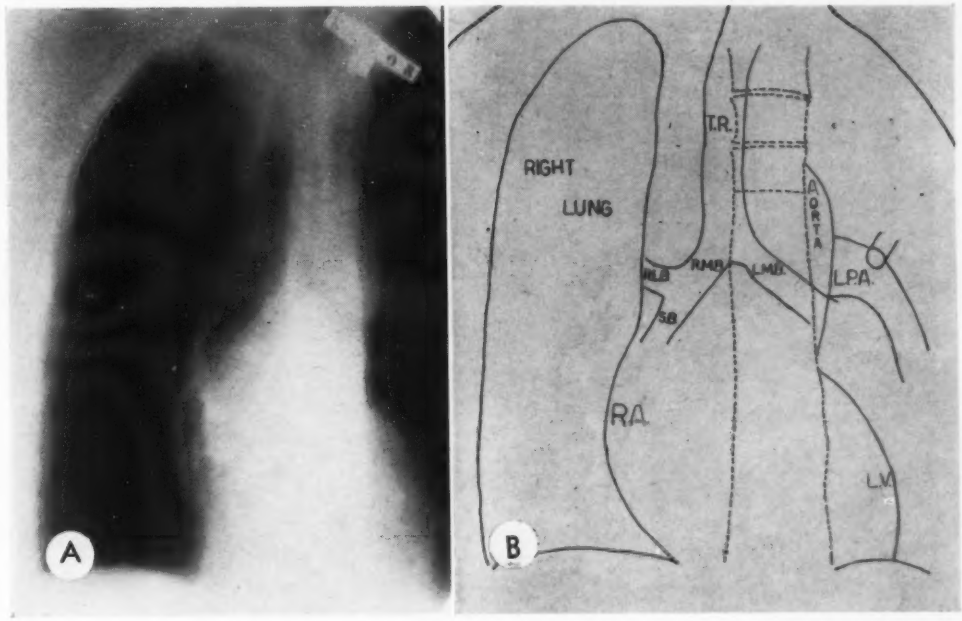

Fig. 6. (*A*) Posteroanterior laminagram at 8 cm. from the posterior plane in erect position and in full inspiration. Distance 1.5 meters. Note that the mediastinal shadow moved to the left and almost reached the midline. The right superior lobe bronchus displayed a somewhat abnormal appearance which is not seen in the reproduction. The right pulmonary artery is not seen, but the left artery is clearly shown and is of increased size. (*B*) A drawing of (*A*). LPA, left pulmonary artery; RMB, right main bronchus; LMB, left main bronchus; RLB, right superior lobe bronchus; SB, stem bronchus; RA, right auricle; LV, left ventricle.

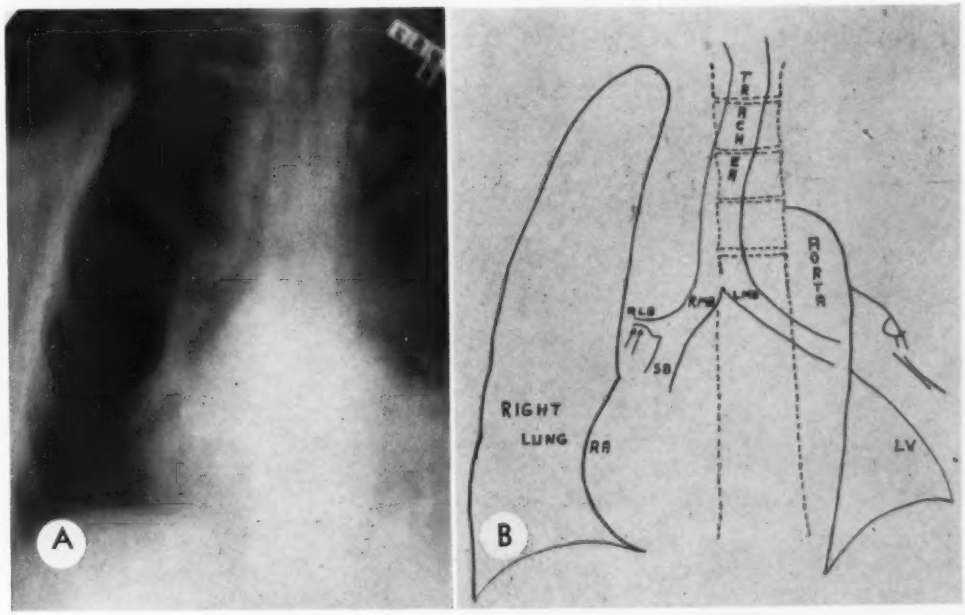

Fig. 7. (*A*) Posteroanterior laminagram at 9 cm. from the posterior plane. Patient in erect position and in full expiration. The mediastinum shifted to the right, and against its shadow it can be clearly seen that the right superior lobe bronchus presents a definite and irregular reduction of its diameter just before branching. (*B*) A drawing of (*A*). LMB, left main bronchus; RMB, right main bronchus; RLB, right superior lobe bronchus; SB, stem bronchus; RA, right auricle; LV, left ventricle.

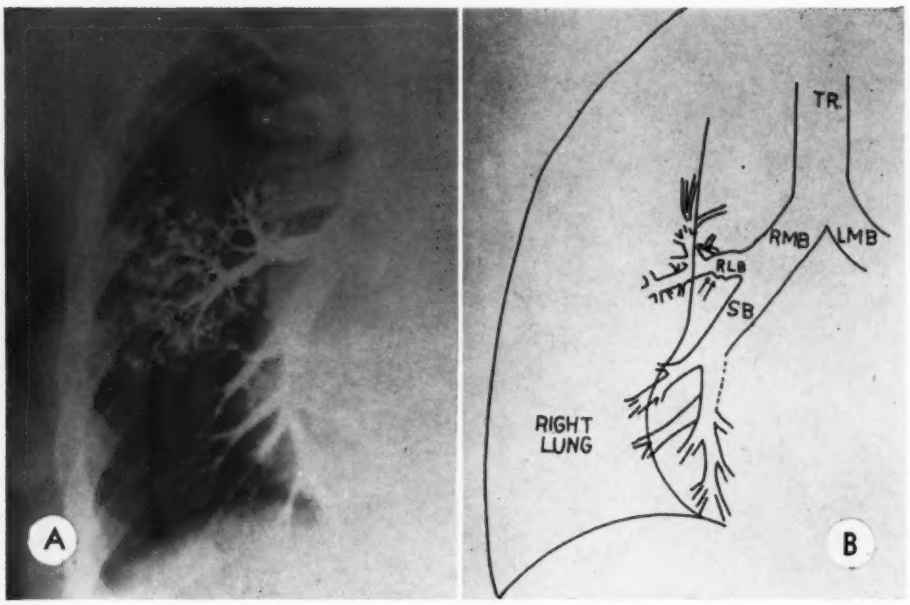

Fig. 8. (*A*) Bronchogram. The right superior lobe bronchus shows a distinct irregularity of its walls just before branching. (*B*) A drawing of (*A*). TR, trachea; LMB, left main bronchus; RMB, right main bronchus; RLB right superior lobe bronchus. SB, stem bronchus.

scribed in our present report. Since in our case there is a right lung, this right lung, of course, must receive its blood supply from the systemic circulation, directly or indirectly from the aorta. It is in this broad sense that we say that it must receive blood from the bronchial arteries. Absence of one pulmonary artery has been found in lung agenesis; in lung hypogenesis, usually with many gross abnormalities in the respective

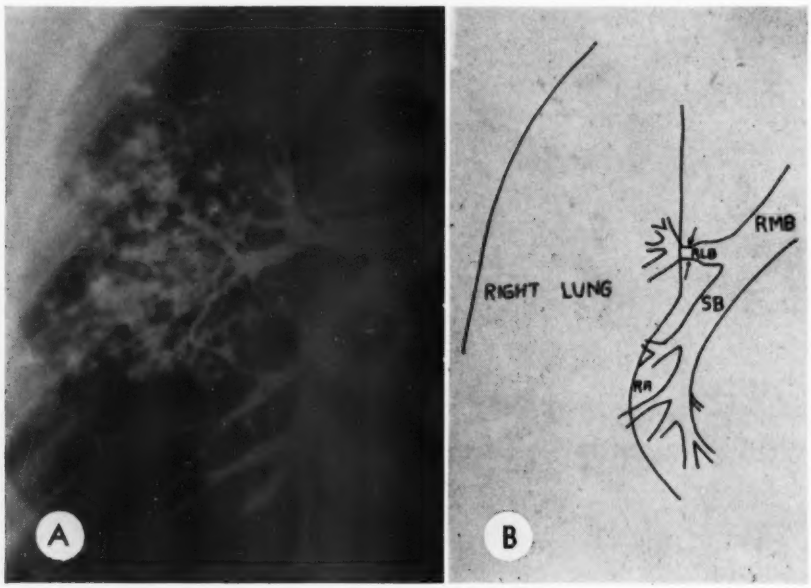

Fig. 9. (*A*) Another bronchogram taken several minutes after the one shown in Figure 8. The irregular appearance of the right superior lobe bronchus persists. (*B*) A drawing of (*A*). RMB, right main bronchus; RLM, right superior lobe bronchus; SB, stem bronchus; RA, right auricle.

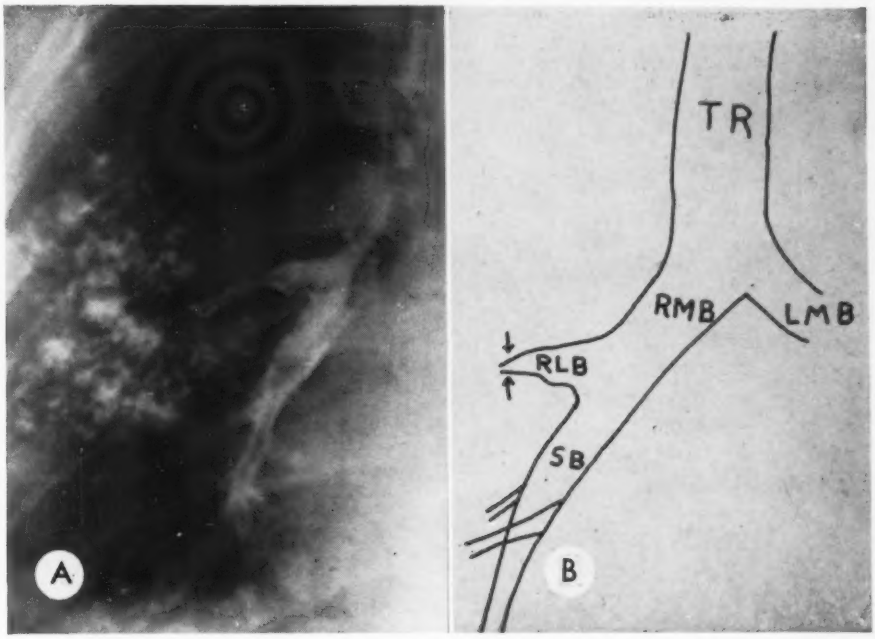

Fig. 10. (*A*) Bronchogram taken after almost complete emptying of the tracheobronchial tree and in full expiration. Tapering right superior lobe bronchus. The roentgenoscopic examination and this bronchogram justify the conclusion that the walls of the right lobe bronchus are pliable in the place where its diameter is reduced (arrows). (*B*) A drawing of (*A*). LMB, left main bronchus; RMB, right main bronchus; RLB, right superior lobe bronchus; SB, stem bronchus.

bronchial subdivisions and also with other severe cardiovascular abnormalities, commonly of the cyanotic type.[5,6,13,16] It has been found, too, in acquired pulmonary non-neoplastic disease during angiocardiography.[9] It has been reported in cases of secondary invasion of a pulmonary artery by neoplasm.[3,9] In the reported cases of neoplastic invasion of a pulmonary artery, besides the other usual evidences of a lung neoplasm, in the angiocardiograms part of the invaded artery commonly persists. We wish to point out that our patient, J. B. F., is a tall, vig-

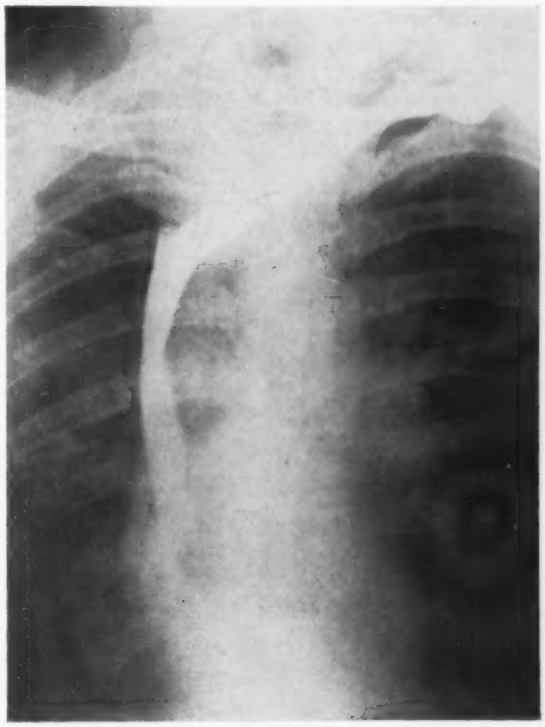

Fig. 11. Angiocardiogram taken two seconds after the beginning of the injection. The right cardiovascular contour is formed by the superior vena cava and right auricle. The right auricle is filled and the right ventricle is beginning to fill. No contrast medium in the pulmonary artery (obtained in erect position at a distance of 1.5 meters).

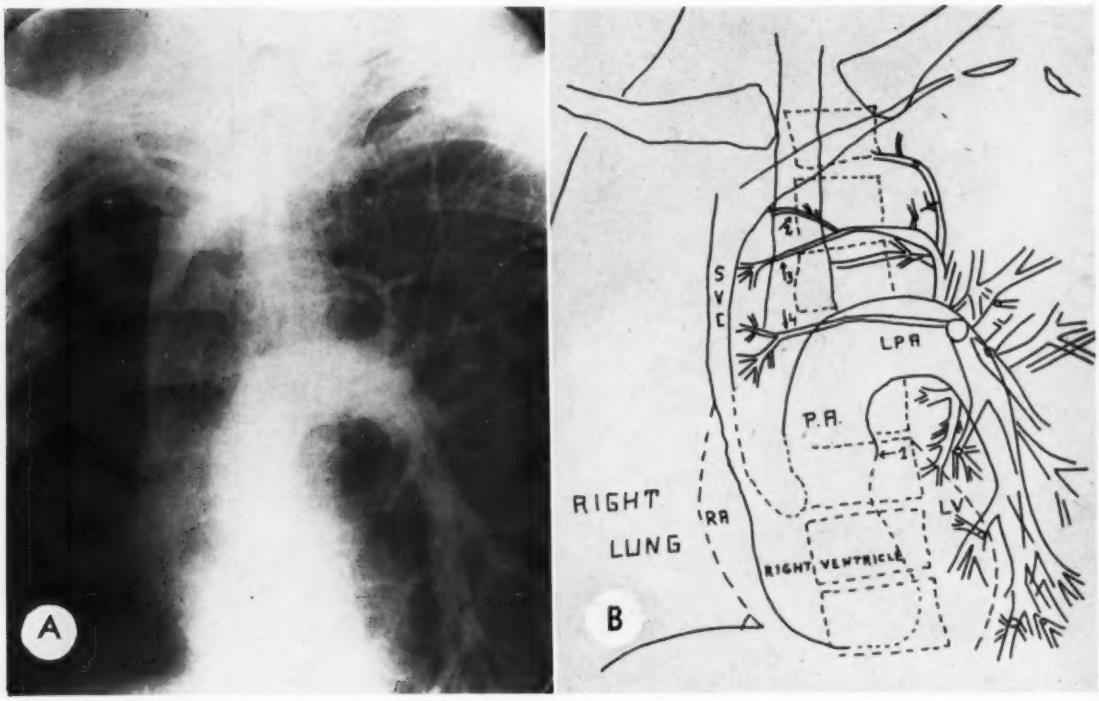

FIG. 12. (*A*) Angiocardiogram taken about one and one half seconds after Figure 11. There are only vestiges of contrast medium in the superior vena cava and right auricle. The right ventricle is faintly outlined because it has emptied almost the whole contrast material into the huge pulmonary artery trunk which is clearly shown. It is even possible to see the bulging of the left pulmonary sinus (arrow *1*). The pulmonary trunk arches to the left forming the left pulmonary artery. Its contour is very precise. There is no sign of the right pulmonary artery. At the left the contrast medium oulines the pulmonary markings in their minute details. Note that from the left pulmonary artery some important vessels (arrows *2*, *3* and *4*) run to the right and so we can gain an idea of the medial limit of the left lung which invades the mediastinal space occupying part of the place left by the hypoplastic right lung. There is no trace of contrast medium in the right lung. (*B*) A drawing of (*A*). SVC, superior vena cava; RA, right auricle; PA, pulmonary artery; LPA, left pulmonary artery; LV, left ventricle.

orous and apparently healthy man and he has had many medical examinations according to the routine in the Brazilian Air Force Medical Service, each one including at least one roentgenogram (abreugraphy); that from the standpoint of standard roentgen interpretation this is a case of *dextrocardia* or *dextroversion*[6,10,12,15] due to the absence of the right pulmonary artery and consequent right lung hypoplasia; that despite the right lung hypoplasia and its lack of function, as far as hematosis is concerned, the right bronchial tree appeared practically normal not only to us, judging from the bronchograms, but also to such an experienced bronchoscopist as Dr. Hollinger.

SUMMARY

The absence of the right pulmonary artery in an apparently healthy man is reported. No similar case has been found in the available literature. The great interest in cardiac and great vessel anomalies has led many experienced authors to report their findings and we found no mention in their books and articles of a similar case.[1,2,6,7,9,11,14,16,17] From the consulted references we chose chiefly those which specifically concerned pulmonary artery anomalies. In the routine roentgenograms we cannot see the right hilus and we can easily see that the left pulmonary artery is much larger than normal. Although these facts are hardly significant in an apparently nor-

mal patient they could give a clue to the diagnosis, but to the authors and to many distinguished radiologists who have seen this case, the definite diagnosis was established only after seeing the angiocardiograms.

We believe it would be correct to include this case among those called dextrocardia in a broad sense,[15] Type III,[10] due to absence of the right pulmonary artery.

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ADDENDUM

After having submitted this paper for publication, we found an article by I. M. Madoff and others in the *New England Journal of Medicine*, 1952, *247*, 149–157. They report on the diagnosis of congenital absence of the right pulmonary artery in the presence of aerated lungs made by angiocardiography in a fifteen year old girl. As in our case there were apparently no other associated congenital cardiovascular defects. The girl was asymptomatic and, similar to our case, there was a narrowing of the right upper lobe bronchus.

Their review showed that this anomaly has been reported 9 times since 1850 associated with anatomic cardiovascular abnormalities usually of a severe nature. All these 9 cases have been diagnosed at autopsy or during an operative procedure (2 cases).

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PHYSIOLOGICAL BRONCHOGRAPHY

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DURING the past thirty-four years, since Jackson published the insufflation method of bronchography,[9] radiologists all over the world have devised numerous modifications of this basic technique in an endeavor to satisfy the ever increasing demands imposed by medical and surgical progress.[6,8,15,17,18,19,21] Despite the unquestioned superiority of today's bronchograms over those performed even a decade ago, the need for improvement persists.

All bronchographic techniques require adequate anesthesia of the upper respiratory tract, instillation of some opaque medium into the bronchi, and distribution of this medium throughout its arborizations. The latter is accomplished by utilization of gravity. This plus a good knowledge of bronchial segmentation, appropriate care and persistence on the part of the radiologist will usually result in good bronchograms. Nevertheless the multiple positions required for proper distribution of the opaque medium by the principle of gravity is a common source of discomfort to patient and radiologist. This feature stimulated the project leading to the refinement submitted herein.

Based on the hypothesis, suggested by a colleague, that deep inspiration might distribute the opaque medium throughout the bronchial tree in a satisfactory manner, the present study was undertaken. Initially a reservoir of lipiodol was created in the right or left main bronchus while the patient was lying horizontally in a supine position and the oil was dispersed by asking the patient to take a deep and forceful inspiration. Visualization of the entire bronchial tree resulted in many patients, but not in all. It was soon found that the opaque medium could be dispersed more uniformly and more consistently if the patient rotated self into a prone position. The resulting increased intrathoracic pressure aided in filling bronchi not previously demonstrated by inspiration alone. In some instances, even after inspiration and rotation, the pectoral portions of bronchial tree were incompletely visualized. This was remedied by requesting the patient to take another deep and forceful inspiration while in the prone position. Thus, by utilizing gravity, deep inspiration and increased intrathoracic pressure, the entire bronchial arborization up to the alveolar level could be demonstrated efficiently and readily in practically all patients. During the entire examination the patient remains in a horizontal position and is therefore comfortable.

METHOD

The basic change introduced by this method lies in the distribution of an opaque medium by utilizing physiological principles. Other features of bronchography, such as postural drainage, anesthesia, or roentgenography may be selected preferentially. The only premedication consists of nembutal gr. $\frac{3}{4}$ (or $1\frac{1}{2}$ gr. in a large patient). A 30 cc. large bore syringe is used with adaptor fitted into one end of a radiopaque 16F urethral catheter. Transnasally, the catheter is inserted into the trachea and the patient is placed horizontally on the roentgenoscopy table in a supine position. Roentgenoscopically the location of the tip of the catheter is checked; it should be in the trachea, 2–3 inches above the carina. The patient is now rotated 45° towards side to be filled, and under roentgenoscopic guidance 15 cc. of lipiodol is instilled rapidly and allowed to gravitate into the main bronchus. In this position the upper lobe

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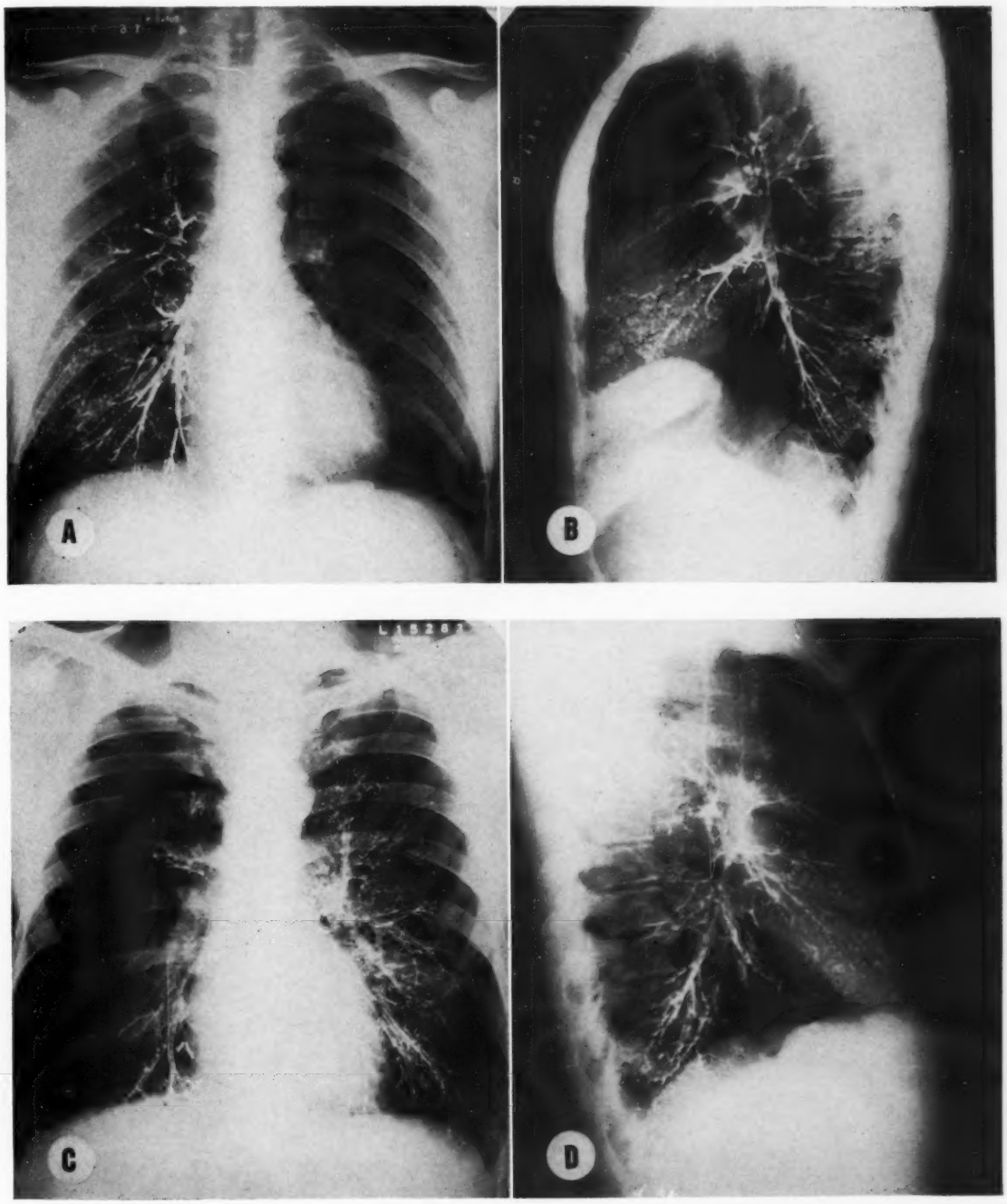

Fig. 1. Complete filling. (*A*) (*B*) Right bronchogram. (*C*) (*D*) Left bronchogram.

bronchus fills readily. The catheter is then withdrawn and the patient is returned to the supine position. The safe light is turned on and the patient is asked to take a single deep breath. Occasionally a second deep inspiration may be necessary if the patient either misunderstands or takes a shallow one. After a brief roentgenoscopic survey of bronchial filling, the patient is requested to roll into a prone position and asked to take another deep inspiration. A final roentgenoscopic survey is

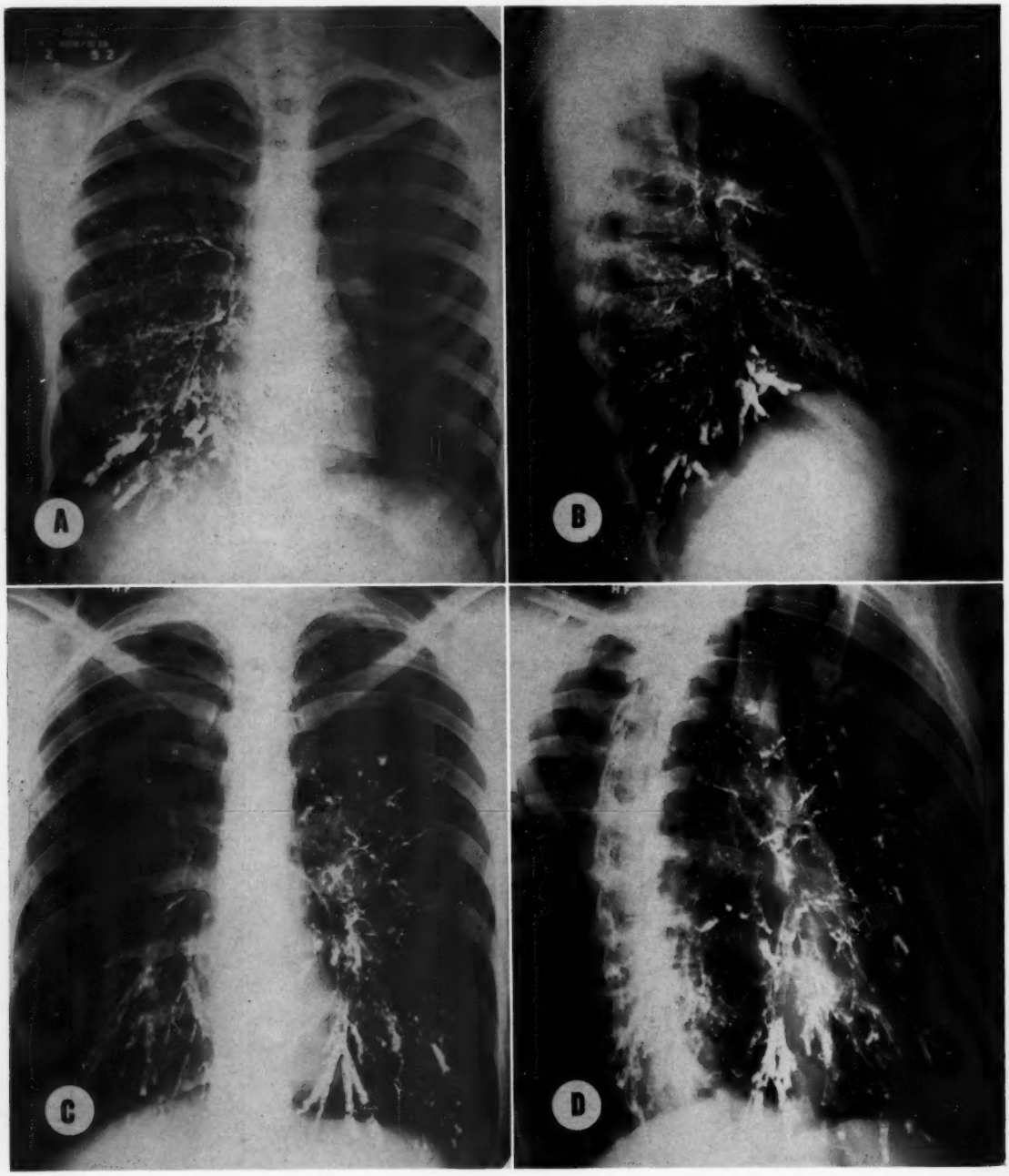

Fig. 2. Bronchiectasis. (*A*) (*B*) Right lower lobe, all basilar segments. (*C*) (*D*) Left lower lobe, all branches, plus the anteromedial division of the lingual.

made and films are taken. Spot films are taken at any stage of procedure.

Based upon experience, it has been found that in most patients the bronchial tree is completely filled with lipiodol after a single deep inspiration while in a supine position; others must also be rotated to a prone position; occasionally an additional deep inspiration in the latter position is also necessary. It is advisable to utilize the complete procedure in all patients with known or suspected pathology.

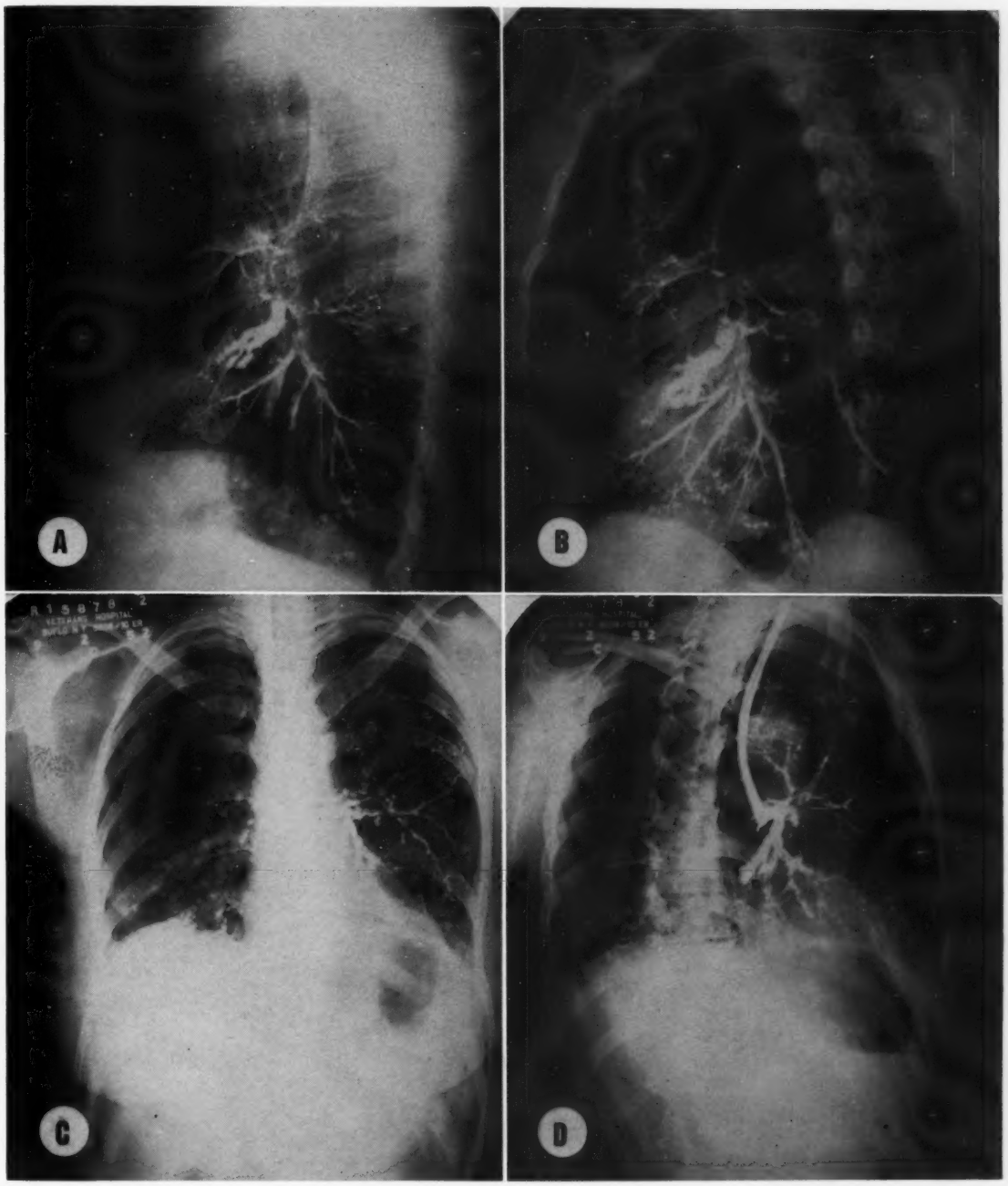

Fig. 3. Bronchiectasis. (*A*) (*B*) Middle lobe, all segments. (*C*) (*D*) Both branches of realigned lingula. Previous left lower lobectomy for bronchiectasis.

DISCUSSION

This study covers 170 bronchographic examinations performed on adults by utilizing outlined physiological principles for the distribution of lipiodol throughout the bronchial tree. In almost all cases, a double contrast outline results demonstrating adequately the trachea, carina, and bronchi up to the alveolar level (Fig. 1). By maintaining the patient in a horizontal position during the entire examination, and by eliminating multiple positions the exam-

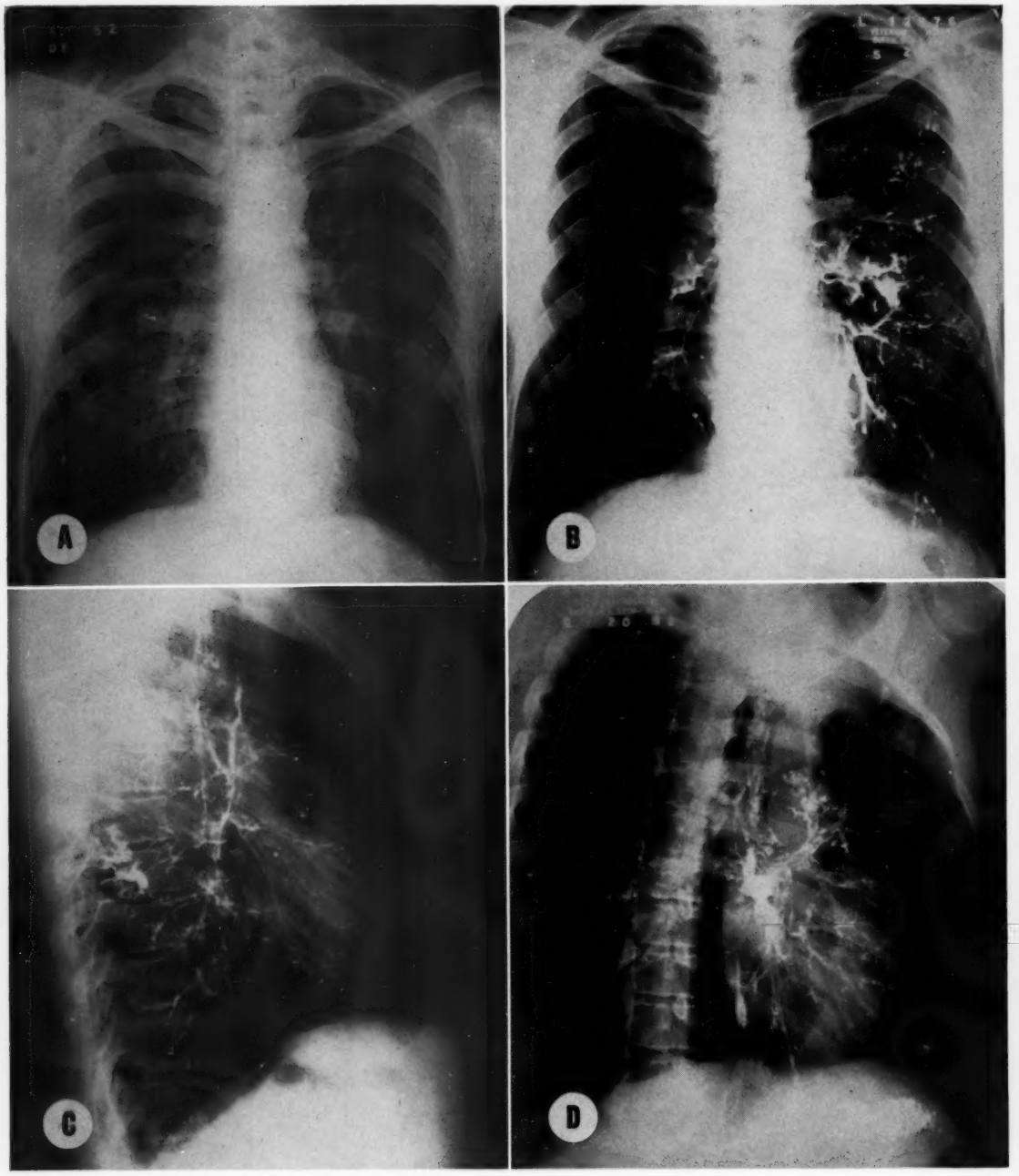

FIG. 4. Lung abscesses, multiple. (*A*) Conventional roentgenogram merely shows distorted lung markings just below the left hilus. (*B*) (*C*) (*D*) Multiple cavities in the dorsal division of the left lower lobe.

ination is expedited, the patient and roentgenologist are comfortable and the overall time is greatly reduced. This method is applicable in debilitated patients and should be applicable in children old enough to cooperate. The technique is easy to master and the patient has no difficulty cooperating. Filling of the middle lobe and lingual bronchi offers no problem.

Because of the efficiency and simplicity of examination, we have utilized physiological bronchography more extensively

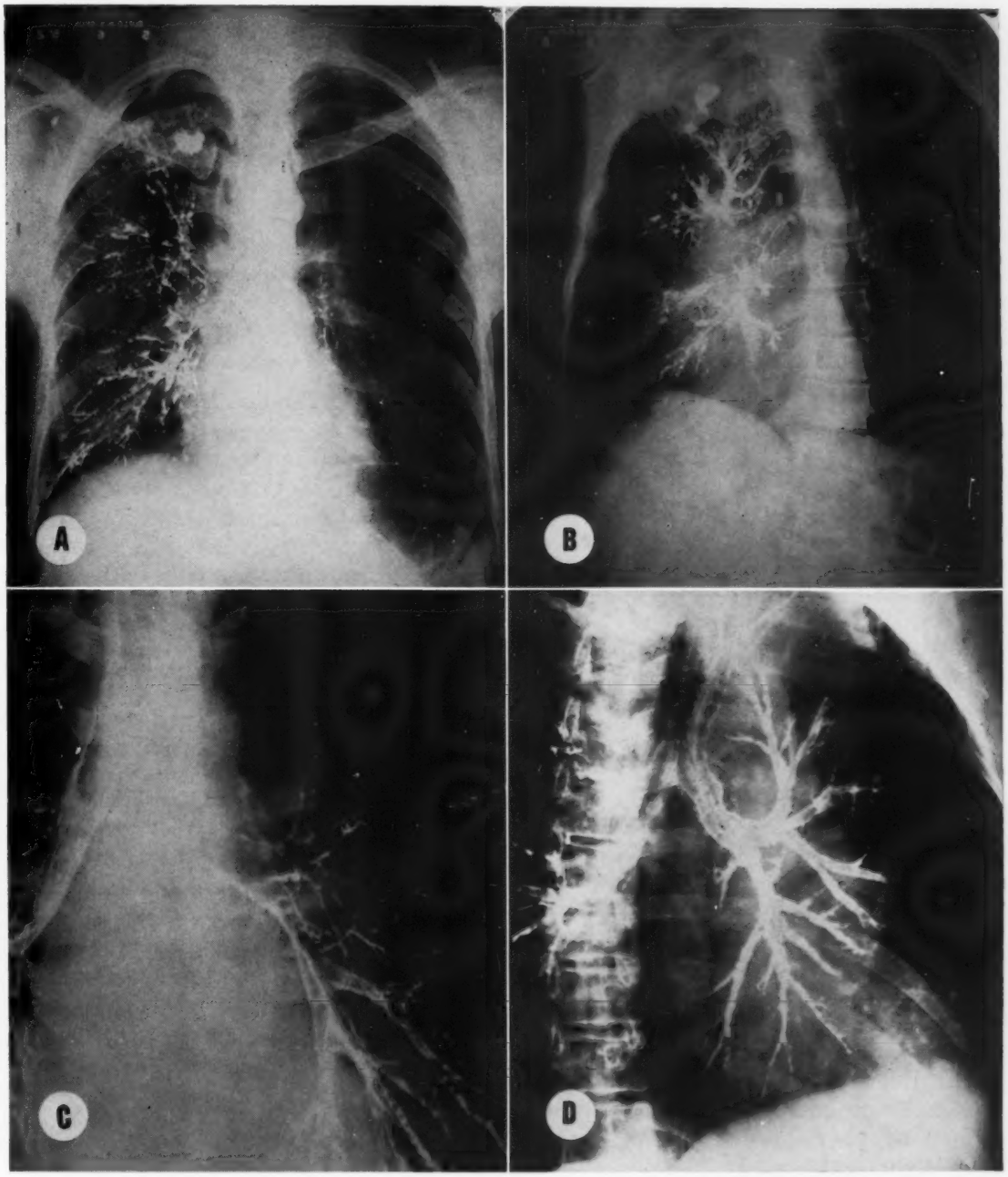

Fig. 5. Shrapnel in right upper lobe. (*A*) (*B*) a Small subdivision of the apical segment leading to shrapnel shows minimal bronchiectasis (confirmed at operation). (*C*) (*D*) Broncholith (calcified bronchial lymph node) of left hilus; the cause of intermittent hemoptysis.

than before. The amount of twenty-four hour residue is similar to that obtained by other methods of bronchography with the exception that flooding of dependent portions of a lung will not occur.

Early in the development of this technique, incomplete visualization of segments or subsegments proved a problem in interpretation. We proceeded on the hypothesis that in bronchographic examinations per-

TABLE I

RESULTS IN PHYSIOLOGICAL BRONCHOGRAPHY
170 cases

Complete Filling		Incomplete Filling
Left—82	Right—76	12
158		

TABLE II

PATHOLOGY IN BRONCHOGRAMS

Bronchiectasis	47	Pneumonia	6
Bronchogenic carcinoma	9	Acute 2	
Lung abscess	8	Chronic 1	
Recent 6		Residuals 3	
Old 2		Cyst	1
Emphysema	4	Diaphragmatic hernia	1
Bronchial relocation, postoperative	4	Calcified hilar lymph node	1
Pulmonary nodule	2	Loculated pneumothorax	1
		Bronchial spasm	1

formed by this technique, portions of the bronchial tree not outlined by lipiodol should be attributable to impaired function of the lung secondary to pathology or to temporarily altered physiology. The latter, it was believed, could probably be inferred in most instances from a negative roentgenogram and/or by the presence of normal spatial relationship of the bronchi. During the interval when inspiration alone was used, about 10 per cent of patients showed segmental or subsegmental nonfilling not attributable to pathology. Since the introduction of rotation and repeat inspiration, the bronchial tree has been completely filled in practically all patients. The problem now is one of evaluating nonfilling due to existing pathology (Table 1). The 12 cases with incomplete filling occurred early in the development of this technique. Seven were attributed to coughing, 2 to technical reasons, and 3 were unexplained. Results in the last 125 examinations were consistently satisfactory and nonfilling was encountered only in pathological bronchi.

The role of pathology in nonfilling of bronchial segments in bronchography performed by physiological principles is incompletely understood. The chief difficulty experienced in interpretation lies in answering the question whether nonfilling is attributable to technique or to pathology, or perhaps to both. It is hoped that experience will clarify their relative roles. In this series of 170 bronchograms, a variety of pathological processes was observed in 88 patients (Table II). With the exception of carcinoma, pulmonary nodules, and pneumonias, evaluation has proved simple.

Bronchiectasis with associated atelectasis, emphysema, and contraction (Fig. 2 and 3); lung abscesses or cavities with or without proximal bronchial ectasia (Fig. 4); postoperative relocation of bronchi are readily demonstrated. Odd cases of lung, bronchial, and pleural pathology in which no difficulty in interpretation was experienced are also included (Fig. 5).

In nonfilling of segments containing pathological processes, bronchogenic carcinoma has provided the greatest problem in interpretation. Several patterns were noted, depending on the location of the neoplasm in relation to the bronchus. In only a few cases was it deemed feasible to express an unequivocal opinion. This subject will be considered in a subsequent paper. Whether physiological bronchography will prove of greater assistance in the definitive diagnosis of chest problems and particularly in bronchogenic carcinoma than other bronchographic techniques can not be predicted without additional experience.

SUMMARY

1. A new technique of bronchography utilizing physiologic distribution of lipiodol throughout the bronchial arborizations by inspiration and rotation of the patient is offered. It is simple, efficient and applicable in most cases.

2. A double contrast outline of the entire bronchial arborization is obtained without inconvenience to the patient.

3. Pathological processes are readily demonstrated, and the interpretation of bronchograms, except in bronchogenic carcinoma, is reliable.

4. The role of physiological bronchography in the diagnosis of bronchogenic carcinoma has not been ascertained, but it is our impression that further investigation is warranted since in several instances the diagnosis was readily made solely from the bronchogram.

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INTUSSUSCEPTION OF APPENDICEAL MUCOCELES

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INTUSSUSCEPTION of the appendix is not a common occurrence. Since the first published case of McKidd[8] in 1859, less than 100 authenticated examples were found in a review of the literature undertaken as recently as 1951.[16] On the other hand, while of relatively frequent occurrence, little has been written concerning the roentgenologic aspects of appendiceal mucoceles.[3] The combination of these two is quite rare. A survey of the literature uncovered only 14 instances of an appendiceal mucocele intussuscepting into the cecum[1,2,4,5,6,7,9,11–16] to which we are presently adding 2 more cases. The first 2 cases were reported by Maclennan[9] in 1919.

CASE REPORTS

CASE I. S.L., a white male, aged thirty-nine, was admitted to the Northern Division of the Albert Einstein Medical Center on January 18, 1953, complaining of pain in the abdomen and rectal bleeding. He had been bothered with "indigestion" intermittently for the past several years. This consisted of a dull ache in the epigastrium accompanied by a sense of fullness which was relieved by milk or alkali. One week prior to admission he developed pain in the epigastric region and on the right side of the abdomen, accompanied by bright red blood in his stools. This had diminished by the time of admission.

Physical examination disclosed a well nourished white male who was in no acute distress. Temperature, 97.4° F.; pulse, 80; blood pressure 140/100. The abdomen was soft and no enlarged organs or masses were felt. There was no tenderness, rebound or muscle guarding. The physical examination was otherwise also completely negative, and essential laboratory studies were all within normal limits.

Roentgen studies on January 19 showed a polypoid mucosal pattern in the duodenal bulb. The progress film revealed that the cecal tip failed to fill or to distend to normal caliber. Barium enema examination the following day disclosed a large polypoid tumor in the cecum, approximately 3.5 by 3.5 cm. Intussusception of the cecum about the tumor was noted. There was no reflux into the terminal ileum (Fig. 1).

The patient was subjected to surgery on January 28. It was found that the cecum was very mobile and there was a tumor in its base which was caused by the intussusception of a thickened appendix into the cecal lumen. The intussusception was reduced without difficulty and an appendectomy was done, excising the broad base of the cecum with the appendix.

The pathology report was as follows:

"Gross: At the base of the appendix in the zone of blending with the cecum, the mucosa is roughened. It has a mucoid appearance and tiny, discrete, orange flecks may be seen.

"Microscopic: In the appendix, in its most proximal portion adjacent to the stoma, there is found a chronic granulation tissue involving and replacing the mucosa. It extends to involve the submucosa, but the underlying wall is essentially intact. Just distal to this focus of granulation tissue is a small area occupying the region of the mucosa and underlying submucosa. This portion is made up of mucus which is divided into compartments by delicate fibrous stroma. No remnants of the original epithelial lining of these locules remain. This mucous material apparently exerts pressure on the underlying muscle and at either end appears to be infiltrating the adjacent submucosa, undermining the mucosa.

"Diagnosis: The exact nature of this mucous lesion is not clear. It may represent a very early and localized mucocele. Arguing in favor of this diagnosis is the very slight mucous distention of some of the mucous glands of adjacent appendiceal mucosa. The possibility that the lesion represents an early gelatinous carcinoma of the appendix must be considered, although I doubt that this is the diagnosis. Certainly, no cells which might suggest carcinoma are found. The invasion, which may represent merely a pressure phenomenon, is a point in favor of carcinoma. In any event, it appears as though the entire lesion has been removed."

CASE II. F.N., a white female, aged fifty-five, was admitted to the Northern Division of the Albert Einstein Medical Center on April 10, 1954, complaining of pain in the right lower quadrant of the abdomen. This was griping in nature, coming on particularly after meals and had been present for about five weeks. She had been able to obtain slight relief by placing herself on a soft diet. She had failed to note any evidence of blood in her stools.

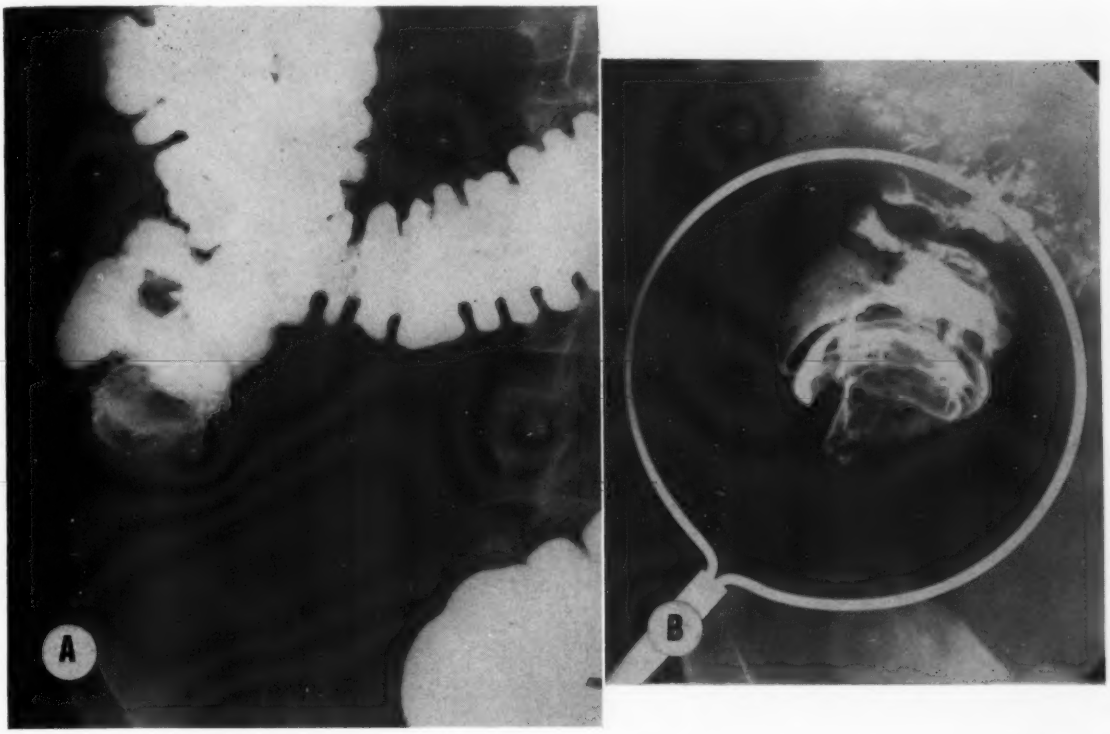

Fig. 1. Case 1. (*A*) Barium enema examination discloses a large, polypoid tumor in the cecum, approximately 3.5 by 3.5 cm. Note failure of appendix or terminal ileum to fill. (*B*) Concentric ring-like shadows of intussusception clearly seen.

Prior to her admission to the hospital, roentgenographic studies of the gastrointestinal tract had been performed in this office. The gallbladder was completely normal and, with the exception of a small hiatus hernia, the upper gastrointestinal tract was similarly free of pathology. Barium enema studies on April 1, 1954, disclosed a large, rounded, cecal filling defect measuring about 2 inches in diameter, which was shown to best advantage on the double contrast study (Fig. 2*A*). Although the cecum appeared to be normal in external contour, ring-like shadows suggestive of intussusception were seen (Fig. 2*B*). The terminal ileum filled satisfactorily but the appendix was not visualized.

Physical examination disclosed a well developed and nourished white female in no acute distress. Temperature, 99.4 F.; pulse, 84; blood pressure, 120/80. The abdomen was slightly protuberant but there was no visible peristalsis, increased muscle tone or rigidity. No organs or tumors were palpated nor was there any localized tenderness. Peristaltic sounds were normal. Laboratory studies were all within normal limits.

Operation was performed on April 14, 1954. Examination of the cecal area revealed a tumor mass arising from the appendix, invaginating itself into the cecum and pushing away some of the layers of the wall of the lower end of the cecum. There was a partial intussusception. The cecum was opened directly over the mass. The mass was reduced from within and it was found to be a large mucocele of the appendix which was pushing aside the layers of the wall of the cecum and pushing the mucosa before it. The meso-appendix was divided between clamps and ligated. There was no communication between the lumen of the appendix and the lumen of the cecum.

The pathology report was as follows:

"The specimen is a longitudinally opened, thick-walled appendix which is dilated and measures 5 cm. in length. It has a circumference of 4 cm. The external surface is pink and shows dilatation of the serosal vessels. The inner surface of the appendix is coated with yellowish material and thick mucinous material.

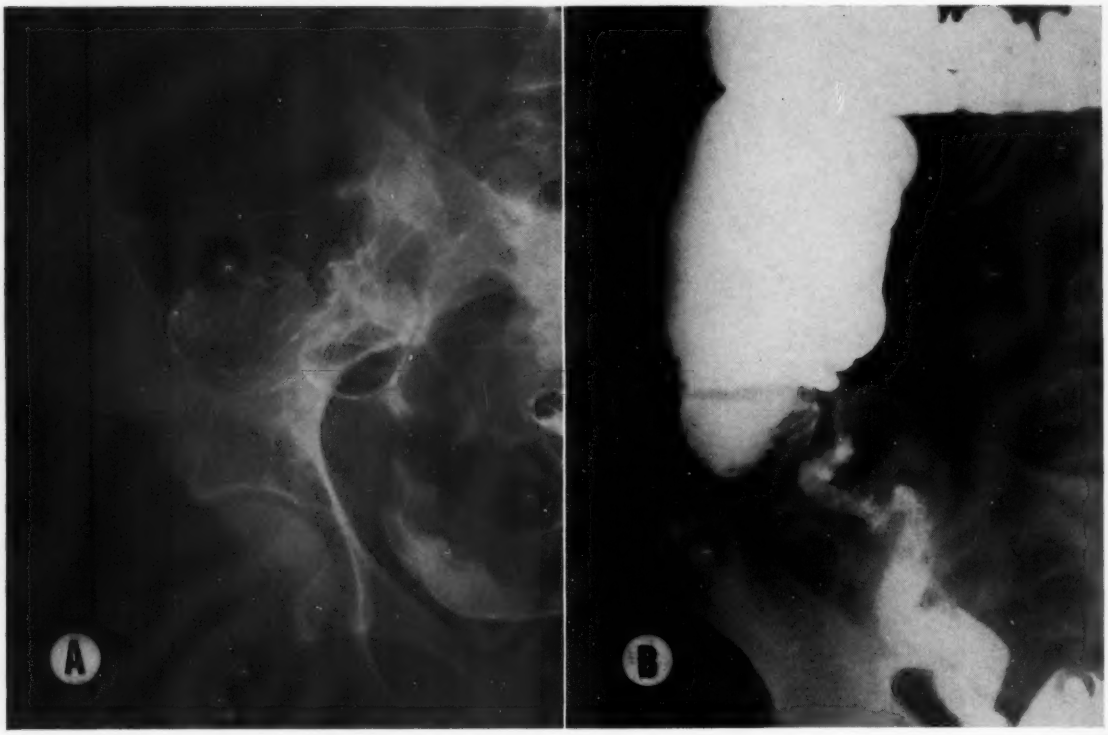

FIG. 2. Case 11. (*A*) The large cecal mass is shown to particular advantage on the double contrast study. (*B*) Concentric ring-like shadows of intussusception demonstrated. In this case, the terminal ileum was visualized and appeared normal, but the appendix, of course, did not fill.

The mucosa is pale, yellowish-gray, alternating with areas of pink. The tip of the appendix is thinner than the remainder, and is divided from the proximal portion by a ridge. Along with the specimen, in the same container, there is a large mass of thick, mucinous material.

"Diagnosis: Mucocele of the appendix."

PATHOLOGY

McSwain[10] has depicted four possible methods of appendiceal intussusception:

1. Invagination of the tip of the appendix.
2. Invagination of the base of the appendix into the cecum (most common).
3. Invagination of the base of the appendix into the tip.
4. Complete inversion of the appendix, which may follow the first or second mechanism, but is impossible after the third.

The second and fourth types may be combined with cecocolic or ileocolic intussusception or both. In addition, intussusception at the site of the stump of a previously removed appendix has been reported several times.

A direct relationship between the development of a mucocele and the so-called normal involution of the appendix was postulated.[12] According to this theory, should involution occur near the base while the tip is still secreting, a mucocele may result. The mucocele-bearing appendix is generally large and there is no communication between the appendiceal and cecal lumens. Marked muscular hypertrophy with injected dilated vessels is usually present and the appendix is filled with a yellow, gelatinous material.

ETIOLOGY

The etiology of the uncomplicated mucocele needs little comment other than the statement that the cause is generally accepted to be obstruction of the appendiceal lumen in the absence of pyogenic infection. Intussusception of the appendix has been

considered secondary to many factors such as hyperactive peristalsis, intestinal helminths, fecaliths, tuberculosis and even trauma. Mucocele has been mentioned by a few writers in addition to those reporting cases. The lack of a meso-appendix has been considered responsible by one writer, and McSwain[10] reported 7 cases in which there was an abnormally mobile cecum, or long mesentery. In children, it has been postulated that enlarged lymphoid follicles might initiate an intussusception. Undoubtedly, the process may stem from an appendiceal stump.

SYMPTOMATOLOGY AND PHYSICAL FINDINGS

Although simple intussusception of the appendix is more common in children, the variety complicating a mucocele occurs exclusively in adult life and is probably related to the afore-mentioned involutional changes. A long history, which in one case covered a nine-year period,[10] is characteristic. The symptoms are those of cramp-like paroxysmal, intermittent pain, which is completely relieved at intervals. The pain is generally in the periumbilical and lower abdominal regions and is usually accompanied by nausea and vomiting, although this was conspicuously lacking in our 2 cases. About one-quarter of the cases may be expected to show evidence of melena, but otherwise bowel changes are not the rule. Physical examination may disclose slight resistance in the appendiceal region, although significant tenderness, rebound or muscle guarding are invariably absent. The temperature, pulse and respiration are usually within normal limits, or at most slightly elevated. Of course, if necrosis or gangrene has supervened[13] the physical findings will accordingly be greater. Distention is infrequent. A mass may be palpable, depending upon the size of the mucocele or the extent of the intussusception, which, in a few instances, has reached the hepatic flexure or slightly beyond.[10,11,14]

The differentiation from acute appendicitis need not be difficult since the symptomatology in intussuscepted mucocele is intermittent and of long standing. The pain is more like that of intestinal obstruction rather than the localized, steady, right lower quadrant pain of acute appendicitis. Physical findings, fever and tachycardia are more common with the acute appendix, but a palpable right lower quadrant mass should be more suggestive of an intussuscepted mucocele.

ROENTGEN FINDINGS

Euphrat,[3] in discussing uncomplicated appendiceal mucocele, claims it should be diagnosed more often by the roentgen ray. If the size is sufficient, displacement and deformity of the cecum may occur. Occasionally, calcification will be detected in the mucocele itself. When the mucocele intussuscepts, added roentgen features of a cecal mass (Fig. 2*A*) and ring-like shadows (Fig. 2*B*) may be apparent. The appendix, of course, will not visualize since there is generally no communication between its lumen and that of the cecum. It is helpful to visualize the terminal ileum and exclude it as the origin of the intussusception.

Unfortunately, many of these cases will not have barium enema examinations. Despite the points differentiating this condition from acute appendicitis, the surgeon seeing the patient in an attack cannot be criticized for suspecting the latter diagnosis.

Should the intussusception be extensive enough to block the ileocecal valve, then the findings of intestinal obstruction will be present. If the barium enema examination is made between acute exacerbations, then a normal study may be expected.

DISCUSSION

The diagnosis of intussusception of an appendiceal mucocele will continue to be made very infrequently preoperatively because of its extremely uncommon occurrence. However, a long-standing history in an adult of intermittent cramp-like abdominal pain, with complete relief at intervals and with few or no physical findings, when fortuitously accompanied by certain roentgen findings, should at least suggest

the possibility of such a diagnosis.

The roentgen findings which may be encountered are mainly two: a mass in the cecum and evidence of intussusception rings. Other important findings, when present with the above signs, are failure to visualize the appendix (although this by itself is of no importance) and filling of the terminal ileum, thus excluding this structure as the intussusceptum. Our Case 11 (Fig. 2*A* and *B*) demonstrated all of these changes.

Despite the somewhat characteristic symptoms and findings, when there is roentgen evidence of a cecal mass, the likelihood of carcinoma will probably take precedence, and rightly so. The diagnosis of intussuscepted appendiceal mucocele will continue to be made at the operating table.

CONCLUSIONS

1. A review of the literature has been made and fourteen cases of intussuscepted appendiceal mucocele have been found. Two additional cases are herewith reported.

2. This is a rare combination of lesions, which, however, presents rather typical symptoms and findings.

3. The roentgenologic manifestations have been presented and discussed.

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SPLENIC VENOGRAPHY*

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DUE to its relative anatomical inaccessibility, the portal circulation has not been widely explored during life by contrast roentgenography. The direct intraportal injection of opaque substances has been accomplished by way of the inferior vena cava in patients who have had portacaval anastamoses,[6,8] and injection into dilated periumbilical veins has achieved visualization in the presence of portal obstruction.[4] More recently, a method for the direct transhepatic injection of opaque substance into a branch of the portal vein has been described.[12] For the most part, however, contrast studies of the portal circulation are based largely upon information derived during laparotomy.[2,5,9,10] Abeatici and Campi[1] and others,[3,7,11,13] have employed a technique which provides a simple and a effective means for the roentgenological study of the portal circulation in the intact patient. The method, probably best described as splenic venography, involves the intrasplenic injection of contrast medium following transparietal puncture.

We have employed a modification of this method in 32 patients, and believe the technique and results to be of interest.

TECHNIQUE

A suitable sedative is used. In the roentgen department the patient is placed in a supine position on the roentgenographic table. The splenic area is delimited by clinical examination, and a 15 gauge needle is introduced into the spleen, generally passing through the ninth or tenth intercostal space in the axillary line. The needle is directed medially and posteriorly. The "drop-by-drop" issuance of blood from the needle indicates that its point lies within the spleen. Ten cubic centimeters of normal saline is first injected in order to "wash" the way in preparation for the contrast substance; 40 to 50 cc. of contrast medium (75 per cent diodrast has been used) is then injected rapidly into the spleen. Hand pressure has proved to be sufficient. At the end of the injection films are made—usually five at one second intervals. Following the study, 10 cc. of normal saline is again introduced in an effort to speed the disappearance of opaque substance from the splenic tissues.

FIG. 1. Postmortem study showing the portal system by means of barium sulfate injection mass.

Employed by us in 32 patients, the method has proved to be safe and without serious subjective reactions. Usually the patient feels a slight pain over the splenic area during the introduction of the needle. There is moderate pain encountered during the injection. The absence of a general reaction such as is seen with angiocardiography is noteworthy, and presumably results from the slow entrance of contrast substance into the general circulation. General

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FIG. 2. A normal splenic venogram. Contrast substance may be seen within the parenchyma of the spleen and outlining the portal vein.

anesthesia has been necessary only in the noncooperative patient. A local anesthetic used at the site of needle insertion may be helpful.

DISCUSSION

The following examples illustrate the utility of this method of examination. Figure 1 reveals the portal circulation in a cadaver following the injection of barium sulfate into the splenic vein. It may be compared to Figure 2, a splenic venogram made during life.

FIG. 3. Partial obstruction of the splenic vein due to pancreatic cyst.

FIG. 4. Carcinoma of the head of the pancreas. The portal vein is elevated much as the duodenal loop in such lesions is depressed and widened beneath it.

Cyst of the body of the pancreas may cause partial extrinsic obstruction of the splenic vein, as is illustrated in Figure 3. Carcinoma of the head of the pancreas produces more or less characteristic findings. The portal vein at its origin from the splenic vein is elevated, forming an arch around the tumor mass (Fig. 4). Such an

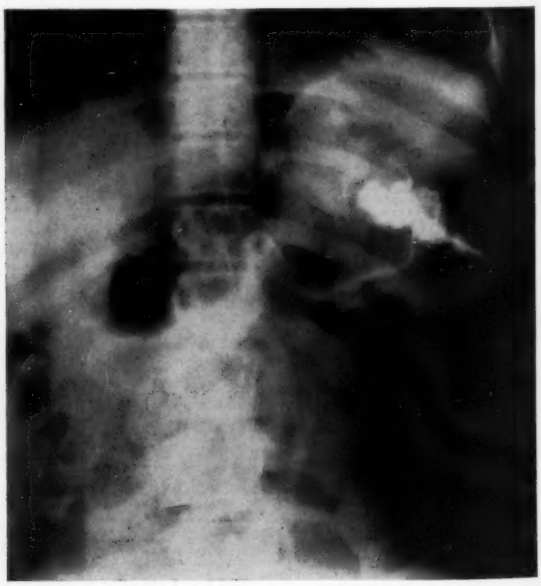

FIG. 5. Thrombosis of the portal vein. The portal vein does not fill; collateral channels (inferior mesenteric vein) are seen.

Fig. 6. A mass in the right lobe of the liver distorts branches of the portal vein in this region.

appearance has been present in 6 cases of carcinoma of the head of the pancreas studied by splenic venography. Thrombosis of the splenic or portal vein may be demonstrated with the method. Figure 5 reveals such an obstruction in a case of portal thrombosis and demonstrates backflow of the opaque substance through the inferior mesenteric vein. Intrahepatic masses may cause distortion of radicles of the portal vein. Figure 6 reveals compression of the right branches of this vessel by a calcified liver cyst.

CONCLUSION

It is felt that splenic venography by the technique described and illustrated above is a safe procedure which may be of considerable aid in the study of abnormalities of the spleen, pancreas, and liver, when these cause changes in the pattern of the splenic vein, the portal vein, or its intrahepatic branches.

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GAS EMBOLUS AS A ROENTGEN SIGN OF FETAL DEATH

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THE first demonstration of intravascular gas as a roentgen sign of fetal death was made by Roberts[3] in 1944. Since that time we have been able to find in the literature available to us only 2 further records of this sign of fetal death (Davidson,[1] 1949 and Kettunen,[2] 1952). The purpose of this communication is to emphasize that this roentgen sign is more common than is generally appreciated and to correlate its appearance with the appearance of other clinical and roentgenological signs. The possible source of this intravascular gas is discussed and its various roentgenological appearances are considered.

MATERIAL EXAMINED

The material investigated consisted of 15 cases of fetal death which were roentgenologically investigated in hospital and private practice during the two years 1951–1953. Case reports are included where the roentgenological features showed either typical or unusual appearances.

ROENTGENOLOGICAL FEATURES

In this series intravascular gas was demonstrated in three main portions of the fetal cardiovascular system: (a) the cardiac organ; (b) the umbilical cord, and (c) the fetal vessels. In the 15 cases analyzed, the features were as follows:

(a) *Intracardiac Gas.* Intracardiac gas was present in all 15 cases. A considerable quantity of gas usually filled the cardiac chambers and the constancy of this feature suggested that gas embolism might indeed be the actual mechanical cause of death by causing cardiac arrest. The outlines of the gas-filled cardiac chambers could usually be seen. The cardiac shadow appeared to be the most valuable site for identifying intravascular gas. Its ease of recognition was proportionate to the quantity of gas present in the heart. In some cases (Fig. 1 and 2) the gas was clearly seen while in others (Fig. 3 and 5) its recognition was more difficult.

(b) *Umbilical Cord.* Gas in the umbilical cord usually formed a long linear streak approximating to the anticipated position of the cord, extending from the placenta into the umbilicus. In some cases, where the appearances suggested gas in the fetal vessels, examination showed the gas to extend outside the fetal shadow and in these cases it was thought to be present in the umbilical cord. We have found no record in the literature of the demonstration of gas in the umbilical cord, although the fact that it appeared five times in this series proves that this finding is not infrequent. The degree and extent of gas occurring in the umbilical cord is significant in this series, as we believe it indicates a possible source and path of intravascular gas. Gas in the umbilical vein was also demonstrated in some cases to extend from the cord into the fetal vessels.

(c) *Intravascular Gas.* Gas could also be demonstrated in other parts of the fetal vascular system, but an anatomical estimate of the actual vessel filled with gas proved to be difficult. Gas was found to be present in the abdominal vessels in 9 out of 15 cases. No gas was demonstrated in the pulmonary or cerebral vessels in any case in this series.

DIFFERENTIAL DIAGNOSIS

Fetal intravascular gas shadows have to be differentiated from: (1) maternal intestinal gas shadows; (2) radiolucent lines produced by fetal subepidermal fat deposits; (3) intrauterine gas.

(1) *Maternal Gas Shadows.* Careful examination of accurately centered supine and lateral films usually enable the ma-

ternal gas shadows to be located outside the uterine shadow. Gas in the maternal small intestine or in the pelvic colon affords the greatest confusion.

(2) *Fetal "Fat Lines."* The adipose fetus usually shows well marked subcutaneous fat lines which are especially clear around the flexed limbs. These fat lines may easily be mistaken for intravascular gas shadows but their parallel nature should enable them to be recognized. Nevertheless, the flexed thigh compressed against the abdominal wall may cause some confusion with intravascular gas in the umbilical vessels.

(3) *Intrauterine and Fetal Intra-abdominal Gas.* Usually intrauterine and fetal intra-abdominal gas is associated with infection and maceration of the fetus. The extension of the gas shadows beyond the normal tissue planes enables this type to be differentiated from intravascular gas. Case IV (Fig. 4) is an example of intrauterine gas associated with infection of the liquor amnii with fetal maceration. In such cases the clinical feature of maternal toxicity, etc., also enables the condition to be differentiated from intravascular gas which is not associated with any marked degree of maternal constitutional disturbance.

Fig. 1. Case I. Right lateral supine film demonstrating gas in the heart (indicated by arrow), and in the umbilical cord (arrows). Spalding's sign is positive.

Fig. 2. Case II. Supine anteroposterior film demonstrating gas in the cardiac shadow (arrow) and in the fetal vessels (arrows). Spalding's sign is negative.

Mention must be made of the absolute necessity of obtaining films free of fetal movements as the least movement may obliterate small quantities of intravascular gas. In doubtful cases we have found that an accurately centered "coned view" of the fetus will often confirm the presence of intravascular gas (Fig. 2).

DISCUSSION

There has been considerable speculation as to the possible source of fetal intravascu-

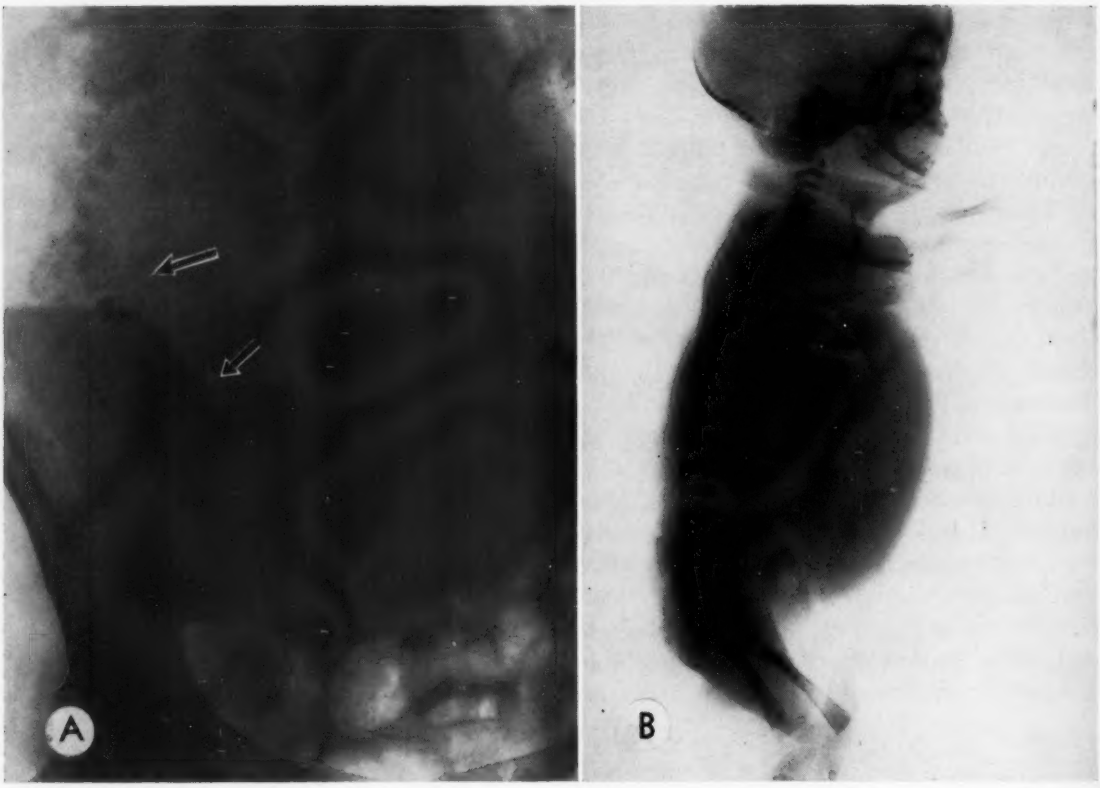

FIG. 3. Case III. (*A*) A supine anteroposterior film demonstrating gas in the heart (lower arrow) and in the fetal vessels (upper arrow). (*B*) Lateral film of fetus. The outlines of the cardiac chamber are only recognizable by the contained gas. Air is also present in the fetal vessels.

lar gas. Both Davidson and Roberts gave the opinion that gas in the fetal circulation is a late sign and arises as a result of "decomposition" of the blood elements.

In our series it is noted that a large volume of gas was seen in the umbilical cord in 5 cases, and in these cases the amount of gas suggested that it was actually being transferred from the fetal side of the placenta into the fetal vascular system. In the 5 cases where a large quantity of gas was present in the umbilical cord films taken at later intervals demonstrated that the quantity of gas in the cord was considerably less, and from this we have surmised that the gas had moved into the fetal vascular tree. It is thus postulated that the gas in the fetal arterial tree is not necessarily the result of "decomposition of the vascular elements" or of the blood components, but rather that it is due to diffusion from the placenta via the umbilical cord into the fetal vascular tree. It is probable that, under certain circumstances, an alteration in the physiological function of the placenta, such as occurs with Rh incompatibility, may result in gas being passed by a differential pressure gradient from the maternal into the fetal circulation. It is possible, in view of the large quantity of gas noted in the cardiac shadow in all these cases, that the actual fetal death may be the result of a gas embolus, analogous to that which occurs in air embolus in the adult.

In one fetus that became available for study, an unsuccessful attempt was made to collect the gas from the fetal heart in an attempt to determine the chemical composition of the gas (Fig. 3*B*).

If the explanation of the source of the gas is accepted then it is readily understood that the appearance of this sign of

fetal death is not necessarily a late one and indeed the gas may actually disappear during the subsequent days after its demonstration.

REPORT OF CASES

Case I. (Fig. 1.) Mrs. L., a primipara aged twenty-seven, in her thirty-seventh week of pregnancy noted an absence of fetal movement for approximately two days before examination. Fetal heart sounds were also not heard. Roentgenological examination, forty-eight hours after fetal movement had ceased, showed negative Spalding's and Hartley's Ball signs, and gas was not visible either in the placenta or any other portion of the vascular tree. A further examination, made eight days later, revealed gas in the umbilical cord, in the vessels and in the fetal heart. Both Spalding's sign and collapse of the spine were now present.

Case II. (Fig. 2.) Mrs. J., a primipara, aged twenty-two, at term had noticed that fetal movements had ceased prior to examination.

Fig. 4. Case IV. A left lateral supine film demonstrating the intrauterine gas. The muscular wall of the uterus can be seen. Some gas is also present in the fetal vascular system.

Fig. 5. Case V. A lateral film demonstrating that gas is present in the cardiac shadow and in some of the fetal vessels (solid arrow). There is no sign of any collapse of the spine and Spalding's sign is negative.

No note of whether or not the fetal heart could be heard was recorded. Roentgenographic examination on June 20, 1952, showed gas in the heart. Some gas was also present in the vessels of lower limbs. There was no gas in the umbilical veins. Spalding's sign was negative, although there was some collapse of the spine. Films repeated two days later still showed an absence of Spalding's sign, but collapse of the fetal spine in a sufficiently marked degree to indicate fetal death was present. Gas was still present in the fetal vessels and in the heart.

Case III. (Fig. 3*A* and *B*.) Mrs. R., a multigravida, aged thirty-three, in the thirty-seventh week of her fifth pregnancy, had noticed that eight days prior to roentgen examination, fetal movement had ceased. Five days prior to roentgen examination the fetal heart sounds were absent. Films taken on June 30, 1952, revealed a vertex presentation with positive Spalding's sign. Gas was noted in the vessels and in the heart shadow itself. Figure 3*B* is a postmortem specimen roentgenogram of the fetus demon-

strating the extensive collection of gas in the heart shadow itself.

CASE IV. (Fig. 4.) Mrs. V., a primipara, aged twenty-seven, in her thirty-seventh week of pregnancy had noticed an absence of fetal movements for seven days prior to roentgen examination. The fetal heart sounds were absent one day prior to roentgen examination. Lateral roentgenograms seven days after cessation of fetal movements demonstrated the presence of gas in the fetus and also some intrauterine gas. This case is illustrated to demonstrate the different appearances produced by intrauterine gas infection and gas in the vascular system.

CASE V. (Fig. 5.) Mrs. D., aged twenty-eight, a multipara, showed a negative Rh factor, with antibodies present, in her thirty-sixth week of pregnancy. Fetal movement had not been noticed for three weeks prior to examination. Films taken revealed a doubtful Spalding's sign and with no evidence of collapse of the spine. Gas was noted in the heart and in the vessels. Films taken seven days later showed that although the fetus had been dead for at least seven days, Spalding's sign and Hartley's Ball sign were both still negative and the intravascular gas had disappeared.

CONCLUSIONS

1. Fifteen cases of fetal death demonstrating intravascular gas noted over a two year period in hospital and private practice are illustrated and discussed.

2. In the authors' experience, intravascular gas is not an infrequent sign of fetal death.

3. It is postulated that gas in the fetal intravascular tree is not necessarily a late sign of fetal death and, in the series under review, occurred five times before the conventional and well known (Spalding's sign and Hartley's Ball sign) roentgenological signs had appeared.

4. In five cases out of fifteen, intravascular gas was the only roentgenological sign of fetal death.

5. It is postulated that gas in the fetus arises not necessarily as a physicochemical change in the blood elements but rather results from a disturbance in the potential gradient between the maternal and the fetal blood pressures.

6. The frequency with which gas is located in the cardiac chamber, itself, suggests that the degree of gas may be sufficient to account for the actual cause of death, *i.e.*, that the fetus actually dies from an intracardiac gas embolus.

7. The possibility of the Rh factor as the cause of the underlying placental change is considered and it is noted that in these series of cases one case showed extensive placental calcification.

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We are indebted to Prof. O. Heyns and to the obstetrical staff of the Queen Victoria Hospital, Johannesburg, Transvaal, for permission to record the hospital cases and also to Drs. D. Black, S. Joel Cohen, M. Orford and E. Rocher for permission to quote cases which have been under their care.

ADDENDUM

Since this paper was accepted for publication two further papers have appeared dealing with the same subject: Gas within foetus indicating foetal death. A. F. Cuck and F. H. Sims. *J. Fac. Radiologists*, 1953, *5*, 112, and Free gas in fetal vessels as a roentgenologic sign of intrauterine fetal death. O. F. Holin. *Acta radiol.*, 1955, *42*, 116.

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Vol. 73, No. 6

VERTEBRAL OSTEOPHYTOSIS*

PATHOLOGIC BASIS OF ITS ROENTGENOLOGY

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IN THEIR classical studies of the human spine published between 1932 and 1939 Schmorl and Junghanns[8,11] observed the occurrence of vertebral osteophytes in 10 per cent of third decade material. From this age group on, these lesions appeared with increasing frequency in up to 90 per cent of all males over fifty years of age and all females over sixty. They recorded a frequent association of vertebral osteophytes with disruption of the intervertebral disc but did not pursue the subject further. In 1934 Shore,[12] following the lead of Knaggs,[9] decribed the same lesion as a special form of spinal arthritis. He named it polyspondylitis marginalis osteophytica. Although Shore's terminology did not become popular, the belief that this osteophytic proliferation, so easily demonstrable on roentgenograms, was a manifestation of arthritis has remained current in most reference books and in almost all diagnostic roentgen reports since that time. The impression has rarely been questioned even though in many cases no evidence of vertebral articular disease or degeneration can be found.

The more recent investigations of Collins[6] indicate that intervertebral disc degeneration begins at an age and occurs with a frequency generally parallel with that of vertebral osteophytes. Collins associated the two lessions inseparably and accepted the osteophyte as a response to disc degeneration. My own histologic studies of an extended series of human vertebrae furnished examples of the development of osteophytes not associated with disc collapse.[2] This observation suggested further examination of the lesion. The following paragraphs and accompanying figures express the results of this investigation.

FIG. 1. Example of early formation of osteophytes at insertion of anterior longitudinal ligament.

Vertebral osteophytosis is a specific tissue reaction to strain which occurs at the areas of insertion of vertebral and paravertebral ligaments into the apophyseal rings of the vertebral bodies.[4] As such, it is in the same category of phenomena as osteophytes protruding from the adductor femoris tubercle, the calcaneus, the pelvis, or from other parts of the skeleton where severe strains occur at the insertions of strong ligaments or tendons. In the vertebrae such strains may accompany a variety of causes ranging from postural imbalance in an obese individual to the late effects of compression fractures.[3] The list of associ-

* Presented at the New York Academy of Medicine, Section on Orthopedics, November, 1953.

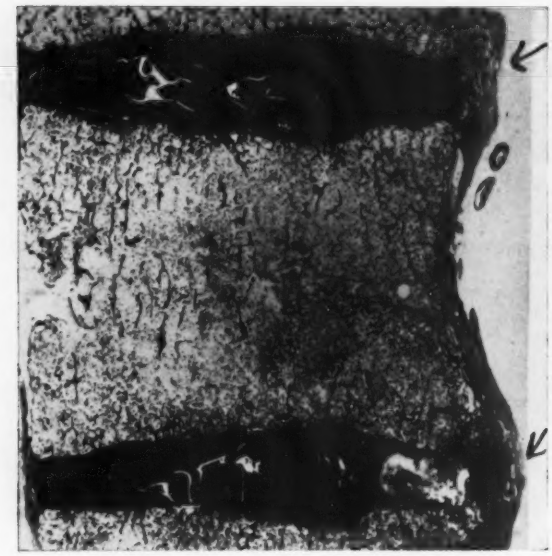

FIG. 2. Further development of osteophytes from adjacent vertebral surfaces along the anterior longitudinal ligament.

ated or causative lesions includes compensatory stresses following degenerative changes within the vertebral disc,[6,7] or within the intervertebral articulations.[1] It may accompany senile osteoporosis.[5] Any of these imbalances or specific lesions may be present without osteophyte formation.

As may be seen in the accompanying photomicrographs osteophytes protrude

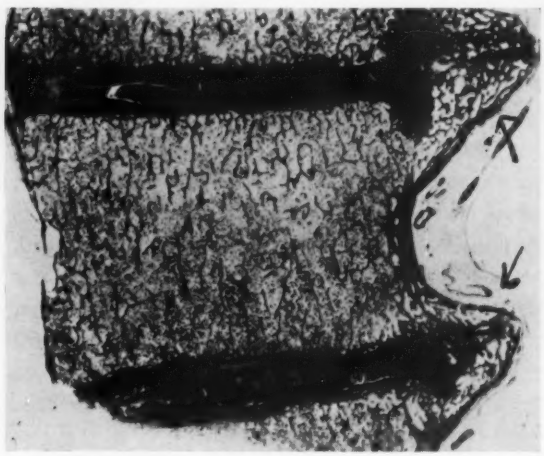

FIG. 3. Complete development of a so-called bony bridge, the final stage of growth of adjacent vertebral osteophytes stimulated by local strain at that level. (Reproduced by permission from *J. Bone & Joint Surg.*, 1952, *34-A*, 110–114.)

FIG. 4. Cervical osteophytes, C_4–5 and C_5–6, involving the anterior longitudinal ligament.

from the chondro-osseous junction at the periphery of the upper or lower surfaces of the vertebral bodies; that is, from the in-

FIG. 5. Cervical osteophytes C_4–5 and C_5–6 of more common type than those shown in Fig. 4.

Fig. 6. Thoracic osteophytosis involving the lateral longitudinal ligaments.

sertions of the intervertebral ligaments at the vertebral ring (Fig. 1 and 2). These lesions differ from the early inflammatory calcification of the same ligaments in rheumatoid or so-called ankylosing spondylitis which ossify only in their later stages.[9,13] The osteophytes infiltrate into the attached ligaments surrounding the intervertebral space. When such osteophytes grow from opposing surfaces they may meet along the connecting ligament and form a bony bridge (Fig. 3).

Fig. 7. Lumbar osteophytes, lumbar spine, late stage, forming bony bridge.

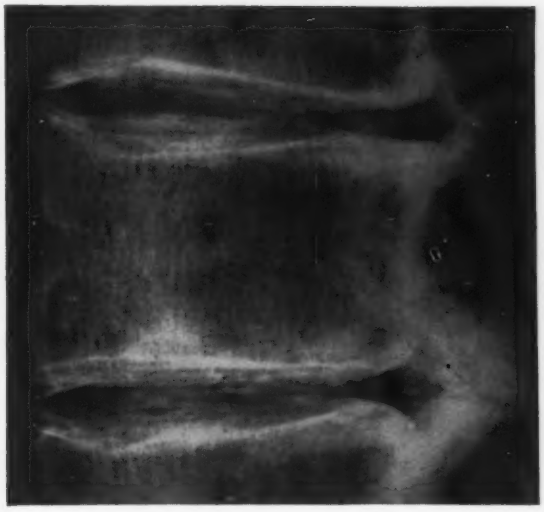

Fig. 8. Lumbar osteophyte, as in Figure 7, from dissected specimen.

In a number of the cases studied, the annular ligament remained intact and no significant areas of degeneration appeared within the space occupied by the nucleus pulposus. There was often no demonstrable loss in the normal distance or change of

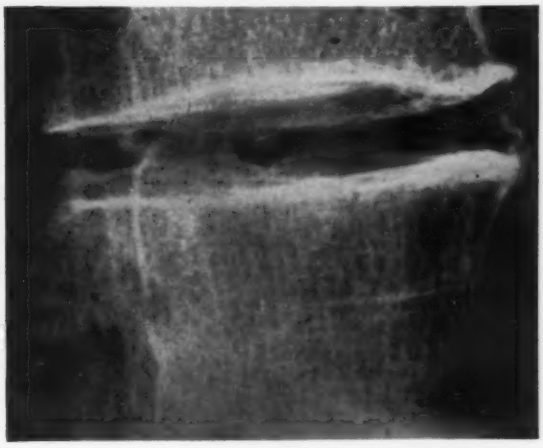

Fig. 9. Early osteophytes from dissected specimen extending across attachments of anterior longitudinal ligament.

contour between the adjacent vertebral surfaces. Histologically these osteophytes differed in no way from similar forms found in the appendicular skeleton. Because they occur in a severely constrained area they are not as expansile as are those formed in more resilient locations. Ordinarily they do not possess a cartilaginous cap. Bone trabeculae are seen emanating from the base of the osteophyte with more mature structure at its denser base and less mature at its tip. The implication of this is the obvious fact that such osteophytes are always larger than the structure visible on the roentgenograms. In the fully developed osteophyte bridging an intervertebral space with its adjacent mate mutual growth pressures create a more homogeneous trabecular density.

The roentgenology of vertebral osteophytosis was studied in dissected specimens and in clinical material. Their characteristics are better demonstrated than described (Fig. 4–9). Osteophytes appear most commonly in the anterior longitudinal ligament, next in frequency along the lateral ligament, and least often in the posterior ligament. They may involve a single vertebra or may be distributed through several or many segments of the spine. In approximately 300 samples of roentgenograms from an unselected series of patients presenting orthopedic complaints, the numerical difference between the occurrence of osteophytosis in the cervical, thoracic, and lumbar areas appeared to be remarkably small, varying with age group, sex, body form, and, of course, associated lesion when present.

Small growths are apt to be asymptomatic incidental findings difficult to relate to any presenting symptom. The development of large osteophytes within the longitudinal ligaments of necessity interferes with their elasticity and therefore with local mobility and the fine mechanism of vertebral balance. This naturally provokes secondary changes in and about the intervertebral articulations and foramina, and a disturbance in the pressure about the nucleus pulposus. Conversely, lesions involving the intervertebral joints or degenerations within the nucleus pulposus may cause abnormal strains on the ligaments, thereby stimulating the formation of osteophytes. In either case pain may occur as the effect of these associated lesions whether primary or secondary. Whether an early uncomplicated vertebral osteophyte can cause pain or not is disputable. The high concentration of pain-sensory nerve endings in these ligaments, experimentally demonstrated by Lindblom[10] would suggest so.

The syndrome of vertebral osteophytosis may remain static, it may progress to include several or even many vertebrae, or it may cause the secondary changes noted above. Although it remains a specific reaction to strain of the intervertebral ligaments, its pathologic prognosis depends upon the associated vertebral changes which it causes or from which it is derived, be these continuous postural strain, compression of vertebra or disc, or spinal arthritis depressing intervertebral articulations. In itself, vertebral osteophytosis is none of these. It is therapeutically misleading to so describe it, and the term should be reserved for cases in which it is manifested without other demonstrable osseous associations.

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THE INDUCTION OF KOEBNER'S PHENOMENON IN PSORIASIS WITH ROENTGEN IRRADIATION*†

By HARRY J. HURLEY, JR., M.D., *and* WALTER B. SHELLEY, M.D., PH.D.
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ALTHOUGH Celsus failed to mention the currently designated "Koebner's phenomenon" in his original description of psoriasis in 35–40 A.D., physicians have come to know it as one of the most disturbing and yet fascinating features of the disease.

In many of these patients almost any form of trauma to the skin will produce psoriatic lesions at the site of the irritation. These include mechanical, thermal and chemical injuries,[3] and even ultraviolet light.[4] Little mention has been made, however, of the effect of the roentgen ray in the production of this phenomenon. Recently a patient was examined at the University Hospital who exhibited such a change, and a report of the case was thought worth while.

CASE REPORT

E. W., a white man, aged seventy-two, was seen initially in March, 1951, because of a pruritic lichenified dermatitis involving the left knee. This patch was irregular in shape and involved a large area of the skin surface of the knee. There were no other sites of involvement and the nails were normal. A diagnosis of lichen simplex chronicus was made on the basis of the clinical appearance of the lesion, and no biopsy was taken.

The patient was given roentgen therapy to the area, which was delivered at 90 kv. (half-value layer 0.7 mm. Al) in doses of 100 r (measured in air) weekly for four weeks to a total of 400 r. A 3.9 cm. cone was used and was placed somewhat eccentrically over the dermatitic patch. Much of the lesion received no direct roentgen radiation, therefore. Under this management alone there was complete clearing of the dermatitis within eight weeks after the last roentgen treatment.

The patient was then symptom-free both objectively and subjectively until September, 1952. At this time he noted the development of a round patch of psoriasiform dermatitis of the left knee (Fig. 1), a scaling fissured area on the right thumb, and dystrophic changes of the nails. The patch of dermatitis on the left knee was exactly 3.9 cm. in diameter and was perfectly round. He insisted he had shown no skin changes on any site during the eighteen month interval between March, 1951, and September, 1952. A clinical diagnosis of psoriasis was confirmed by biopsy of the knee lesion.

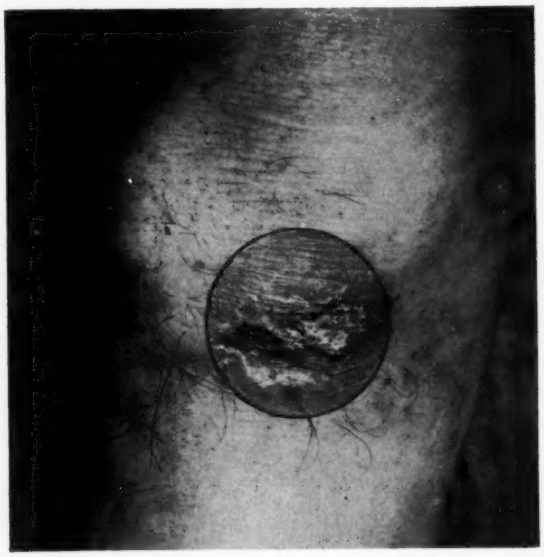

FIG. 1. Appearance of psoriatic lesion on the left knee. The circle outlines the exact site treated with 400 r of superficial roentgen rays eighteen months previously. Note sharp limitation of psoriatic process to this zone.

The psoriatic patch on the knee was thought to be a roentgen irradiation induced Koebner's phenomenon.

DISCUSSION

A survey of the literature reveals a paucity of case reports regarding the appearance of the isomorphic or Koebner-Krebich phenomenon following roentgen

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therapy. Only 2 similar instances of such a process have been recorded.[1,2] Both cases differed in several respects from this one.

In neither case did the interval between the administration of the roentgen radiation and the appearance of the psoriatic lesions exceed four months, while in this instance the response was much more delayed —eighteen months. Furthermore, both of the previously reported cases were receiving deep roentgen therapy for unrelated conditions, one for carcinoma of the cervix[2] and the other for osteoarthritis of the vertebral column.[1] Obviously there was a difference in both the quality and quantity of the roentgen radiation received in these cases as compared with the case reported here. Finally, in each of the other cases, guttate and larger psoriatic lesions developed in the irradiated zones, leaving much of the skin exposed to the roentgen rays free from involvement. In our case it is notable that sharp limitation of the psoriatic process to the treated zone existed, the knee lesion corresponding exactly in size with that of the roentgen-ray cone.

Additional significance may be derived from the delayed response of the patient to the roentgen radiation administered eighteen months previously. It is further proof of the remarkable "memory" of the skin for the occult and apparently innocuous injuries inflicted upon it months or years before.

SUMMARY

A case is presented in which roentgen irradiation was apparently responsible for the appearance of Koebner's phenomenon in psoriasis. The development of this change was exceptional in that it was sharply limited to the irradiated zone and showed an eighteen month delay between the injury and the response.

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TIME-DOSE RELATIONSHIP IN IRRADIATION OF RECURRENT CANCER OF THE BREAST

ISO-EFFECT CURVE AND TUMOR LETHAL DOSE*

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INTRODUCTION

THE purposes of this study are to investigate the optimum tumor lethal dose for recurrent cancer of the breast, and to attempt to express this dose in the form of an iso-effect curve or recovery curve.

An iso-effect curve defines the doses, for different over-all treatment times, which produce similar biological effects. It has two functions. It is a quantitative expression of the summated radiobiologic processes which comprise recovery from irradiation. It serves as a useful guide in clinical radiation therapy. A curve which presumes to measure the recovery process must be based on a scatter diagram wherein the data are collected under experimentally controlled conditions; and the standard error must be relatively small. A curve which serves only as a guide in clinical practice need not be constructed under such rigid rules.

Most of the information concerning tumor lethal dose of cancer of the breast has been derived from the preoperative irradiation of inoperable lesions employing tumor doses ranging from 4,500 to 8,000 roentgens over a period of one to three months. Due to the irregular and changing contours of the breast, there has been considerable inaccuracy in estimating the tumor dose, as well as variation of the tumor dose throughout the breast and axilla.

Cohen[1] analyzed statistically the published data of many authors and constructed an iso-effect curve from them. He found this curve to be similar to an iso-effect curve derived from his own observations on cancer of the breast. However, these clinical data were not collected under controlled conditions. There was marked variation in the size of the tumor, size of the area irradiated, stage of the disease, and nature of the tumor bed. Furthermore, since the patients had only one lesion, it could not be ascertained whether the administered dose was optimal or excessive.

Similarly, the iso-effect curves of Strandqvist[6] for carcinoma of the skin were based on one lesion in each patient. The given doses were dictated predominantly by clinical considerations and not by a predetermined experimental plan. The biologic test object had a wide range of individual variation in radiosensitivity. For example, skin cancers successfully irradiated with a single dose by Strandqvist had tumor lethal doses which ranged from 1,400 to 2,700 r. This wide range of radiosensitivity reduced the accuracy of the final iso-effect curve, not only with respect to the recommended dosage as indicated by the position of the curve in relation to its coordinates, but also the shape and the slope of the curve. The successful doses, from which the major iso-effect curve was derived, had one thing in common: they destroyed the tumor. However, some were minimal tumor lethal doses, and some were excessive. The resultant scatter diagram represented doses larger than optimal, and the iso-effect curves may have been erroneous in shape and position. Strandqvist attempted to overcome this weakness by supplementary comparison of inadequate and excessive doses. The latter type of analysis is not too satisfactory, as the paucity of observations and the widespread dispersion of the points in his scatter diagram did not substantiate the shape of the derived iso-effect curve.

* From the Department of Radiology, New York University College of Medicine, New York. Presented at the Fifty-fourth Annual Meeting, American Roentgen Ray Society, Cincinnati, Ohio, Sept. 29–Oct. 2, 1953.

Nevertheless Strandqvist's final iso-effect curve for skin cancer is a very useful clinical guide. As a measure of the quantitative recovery processes, it is less reliable.

A new experiment was designed in an attempt to overcome these objections. A group of patients were selected who had multiple small nodules of recurrent breast cancer on the chest wall. They lay in a similar tumor bed. The size of the irradiated area was constant.

MATERIAL AND METHODS

Twenty-nine patients, who had been subjected to radical mastectomy for cancer of the breast, subsequently had recurrent subcutaneous nodules on the chest wall. The skin nodules, with few exceptions, ranged in size from 0.5 to 2 cm. in diameter.

One group of 11 patients had multiple lesions, ranging in number from four to twenty-seven in a single individual, and averaging thirteen per patient. There was a total of 142 lesions in this group. Data from this group were analyzed separately, because multiple observations in the same patient permitted corroboration of dosage. As a result, a crude but useful iso-effect curve of tumor lethal dose for each patient's tumor could be constructed. Another group of 18 patients had one, two, or three lesions per patient. There was a total of thirty lesions in this group. In this group, the administered dose could not be compared with other lesions in the same patient.

Each nodule was individually irradiated with 200 kv. roentgen rays (half-value layer 1 mm. Cu) through a circular or square portal whose area was 20 cm^2. All stated doses represented the calculated tumor doses. The total tumor dose to one nodule differed in magnitude and over-all treatment time from that of the other nodules in the same patient.

The Corroborated Dose Technique. The corroborated dose technique was designed to avoid the error of other authors wherein they accepted as an optimum dose any given dose which destroyed the tumor, provided it caused no radionecrosis. In this study, when a certain dose in a specified over-all time was found to be efficient, another lesion would be irradiated with a smaller dose in the same over-all time. This procedure was continued with other nodules until a dose was reached which failed to destroy the tumor. The smallest effective dose was called the "minimum tumor lethal dose," *i.e.*, the smallest amount of radiation within a specified over-all time which caused complete disappearance of a lesion for at least six months. Since this was a clinical study on ambulatory patients, it was often difficult to deliver groups of doses within the same over-all time period, and certain compromises were made, as illustrated in Figure 2.

The corroborated dose technique is best illustrated by the description of a case.

Case 1. This patient had twenty-seven skin or subcutaneous nodules on the anterior chest wall. Each nodule was individually irradiated with a specific dose given in a specific over-all time, and its shrinkage curves recorded. In Figure 1, circles indicate the doses which failed to destroy the tumor. The solid dots indicate successful doses, the small dots representing given doses which were effective but probably excessive in amount, the large dots representing corroborated *minimum tumor lethal doses* because they lie close to the zone of inadequate dosage. For example, 1,700 r in a single dose failed in two instances. There were no one-day doses which were successful. Two lesions were destroyed with 2,160 r in two days, and one lesion with 1,800 r in two days. Therefore, the lesser dose of 1,800 r in two days was considered to be corroborated and was designated as a "minimum tumor lethal dose." It was drawn as a large dot. In this case, three additional time-doses were similarly designated: 2,400 r in six days, and two nodules receiving 3,240 r in fifteen days (Table 1).

These minimal tumor lethal dose points, together with seventeen additional points from six other patients were transferred to a scatter diagram (Fig. 5).

Four patients with multiple lesions failed to yield corroborated dose points.

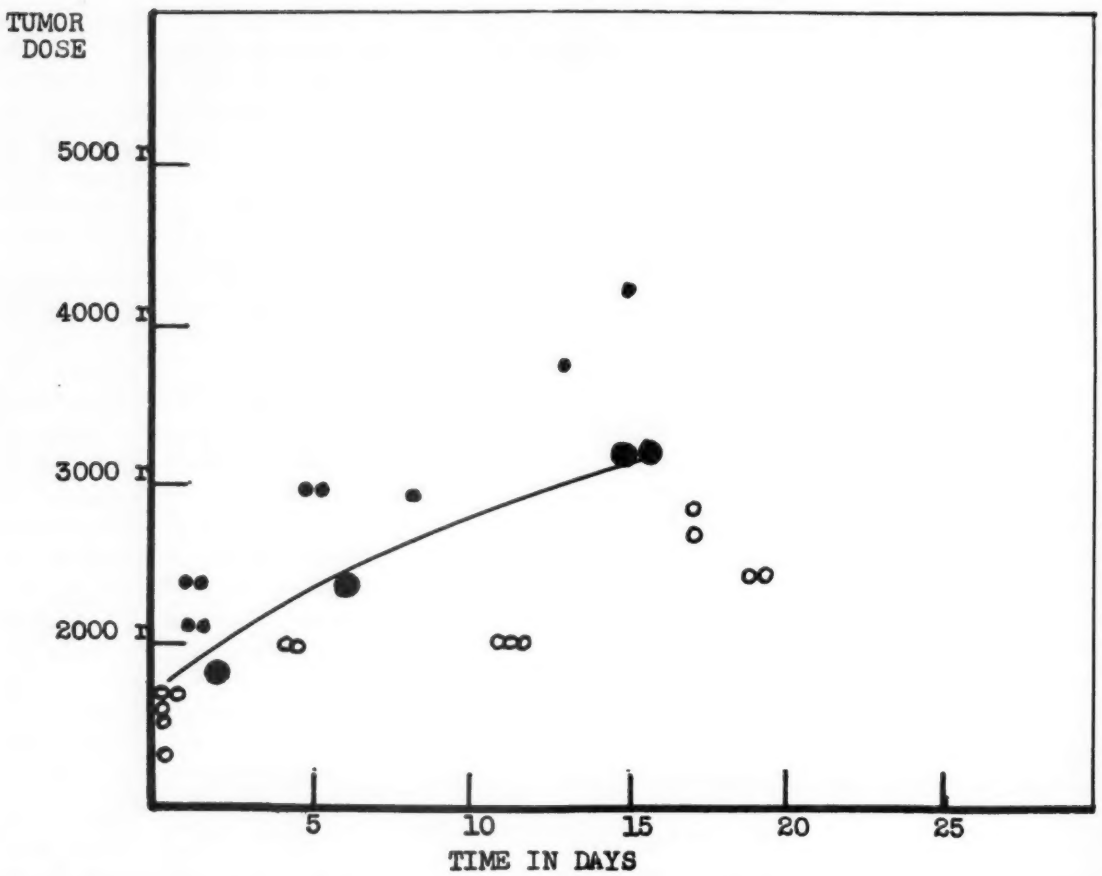

FIG. 1. Case 1. Scatter diagram of doses given to twenty-seven lesions in the same patient. The circles represent failures. The solid dots represent successful doses. The small dots represent given doses which were effective but probably excessive in amount; the large dots represent corroborated minimal tumor lethal doses, because they lie close to the zone of inadequate dosage. The iso-effect curve is based on the latter points, and is drawn freehand.

The Tumor Shrinkage Curve. Although observations of the rate of shrinkage of tumors are inaccurate and consequently statistical deductions are unreliable, nevertheless some useful information is derived from study of tumor shrinkage curves.

The shrinkage of each nodule was plotted as a curve on a graph, and was usually sigmoid in shape. Each observation entailed measuring the three diameters of a tumor and calculating its volume. In this study, the small size of many of the skin nodules did not permit accurate measurement of the diameters. Reliance was placed on a crude estimate as to whether the tumor had shrunk 25 per cent, 50 per cent, 75 per cent, or 100 per cent. Although intermediate observations might be inaccurate; the time of complete disappearance of the tumor was fairly accurate.

As a result of constructing shrinkage curves of thousands of tumors of different types for the past twenty years, certain basic generalities became evident. Extremely radiosensitive tumors, such as certain lymphomas or seminoma, shrink at a rapid rate. Their typical shrinkage curves would lie in the shaded area of Figure 3. Certain other tumors shrink at a slower rate, but finally disappear one to three months after the first treatment. Their shrinkage curves would fall within the unshaded area of Figure 3. Naturally, the slope of these curves would be modified by changing the size of

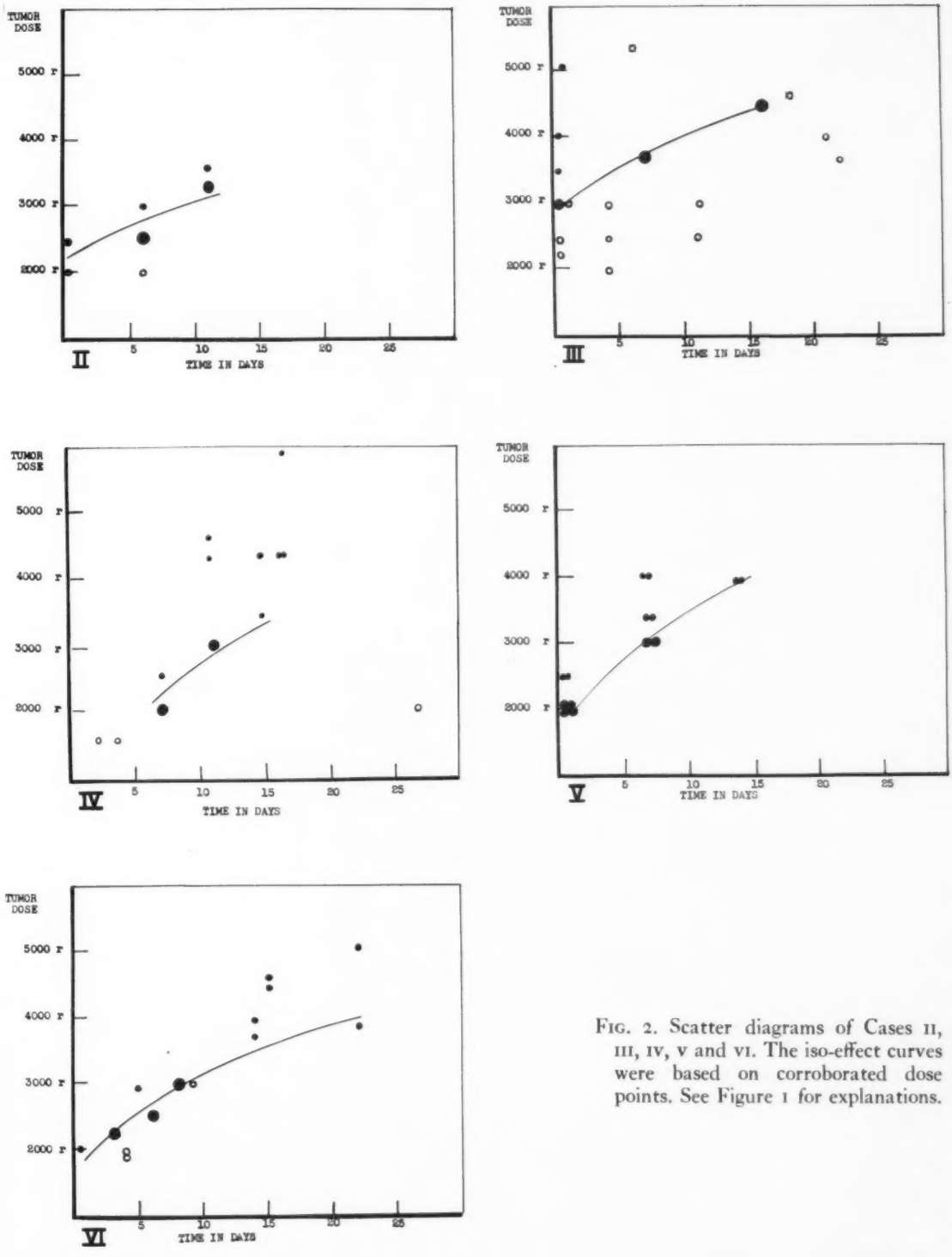

FIG. 2. Scatter diagrams of Cases II, III, IV, V and VI. The iso-effect curves were based on corroborated dose points. See Figure 1 for explanations.

the daily and total tumor dose. The sigmoid curve of the right hand margin of the shaded area is descriptive of a plurality of all types of tumors successfully irradiated. For the purpose of this experiment, it has been adopted as an arbitrary boundary between two zones.

Figure 3 serves only to illustrate all the

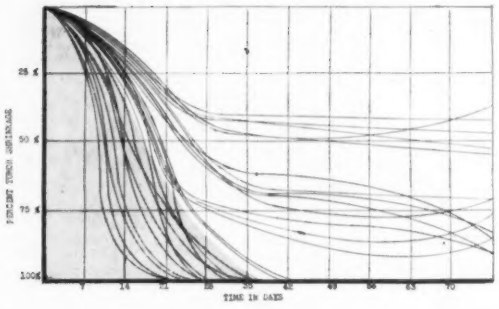

FIG. 3. Case I. Tumor shrinkage curves of the twenty-seven lesions in this patient. The shaded area defines the *zone of efficient shrinkage*. In the unshaded zone there are two curves of "slow but complete shrinkage," and twelve failures.

shrinkage curves of Case I, whose corresponding tumor doses are illustrated in Figure I. It is seen that some shrinkage curves fall within the shaded zone. These were produced mostly by excessive doses. Some

TABLE I

CORROBORATED MINIMUM TUMOR LETHAL DOSES

	Dose (in r)	Over-all Time
Case I	1,800	2
	2,400	6
	2,954	8
	3,240	15
Case II	2,250	3
	2,500	6
	3,000	8
Case III	3,000	1
	3,750	7
	4,425	17
Case IV	2,000	7
	3,360	15
Case V	2,400	6
	3,200	10
Case VI	2,000	1
	2,000	1
	2,000	1
	2,000	1
	3,000	7
	3,000	7
Case VII	4,800	11

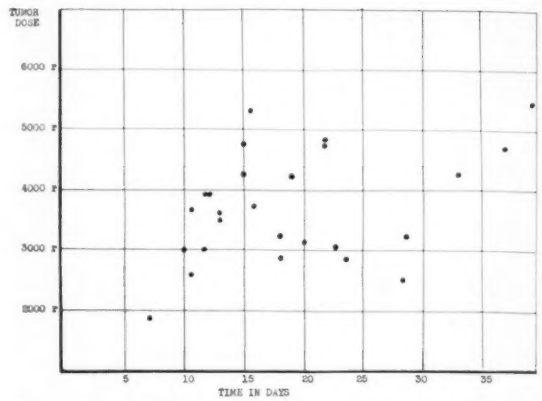

FIG. 4. Scatter diagram of successful tumor lethal doses given to 18 patients with only one or two lesions per patient. Note the wide dispersion of given tumor doses which occurs when there is no opportunity to corroborate one dose with others in the same patient.

shrinkage curves of successfully irradiated lesions lie within the unshaded zones. Many shrinkage curves failed to reach the base line. Their doses were inadequate and are represented as circles in Figure I.

Although the shape of a shrinkage curve is not too reliable an indicator of the effect of a given dose, it was found that curves of *slow but complete shrinkage*, which lay in the unshaded portion of the graph, told a somewhat consistent story, especially when the data were otherwise corroborated. Therefore, data related to these curves will be separately analyzed.

ANALYSIS OF DATA AND CONSTRUCTION OF ISO-EFFECT CURVES

As stated in the introduction, there are two major weaknesses of published iso-effect curves. There is lack of corroboration of the tumor lethal dose level (*i.e.*, whether the given dose was maximum, optimum, or minimum), due to the fact that there was only one lesion in each patient. The broad range of radiosensitivity of the biologic test object introduces a large statistical error so that thousands instead of hundreds of observations would be required to obtain suitable average curves.

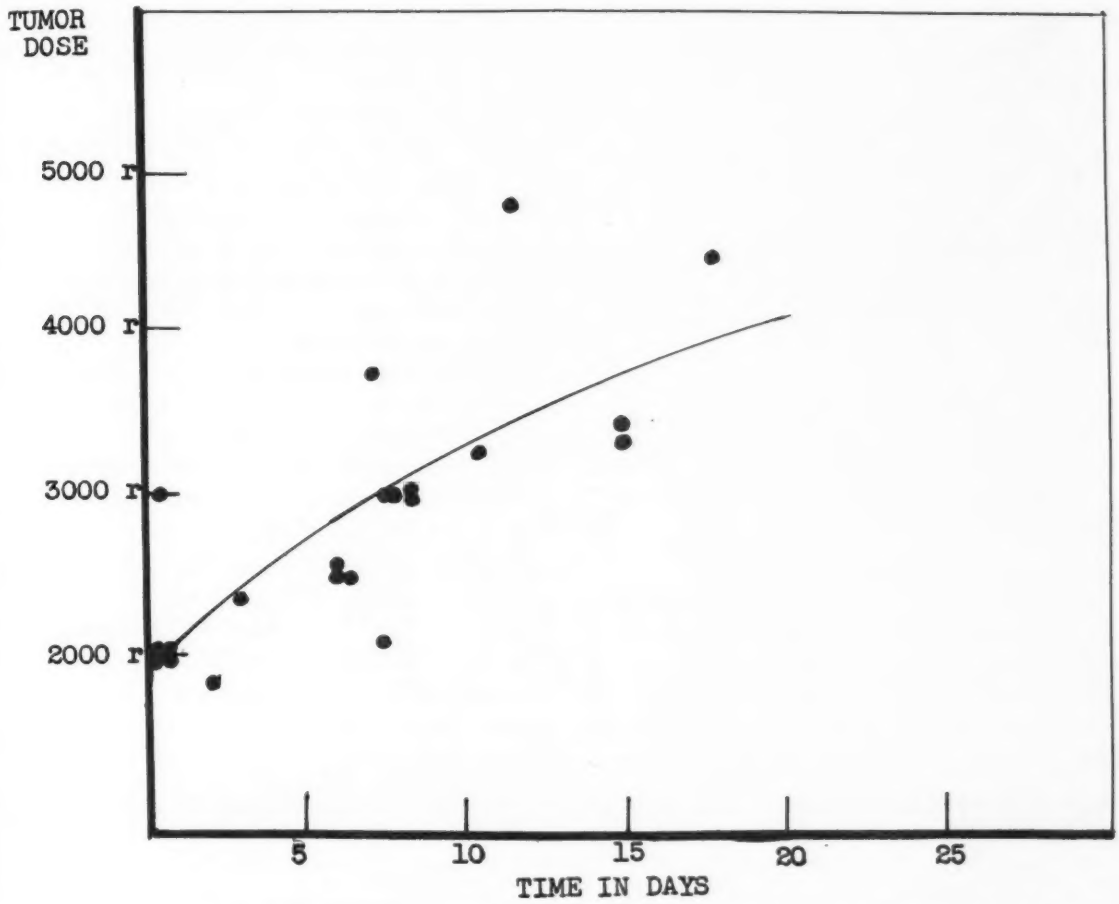

Fig. 5. "Corroborated doses" from 7 patients with multiple lesions enumerated in Table 1. The iso-effect curve was drawn freehand.

Patients with Single Lesions. The difficulties in constructing an iso-effect curve from uncorroborated points is illustrated in Figure 4. The twenty-six lethal dose points were obtained from 18 patients who had only one or two lesions. The given doses were arbitrarily selected on the basis of clinical judgment. The points are widely dispersed throughout the scatter diagram and fail to suggest a trend.

It was impossible to construct a freehand iso-effect curve because the standard error was too great. Thus, when the technique of Strandqvist was applied to breast cancer nodules, we were unable to derive an iso-effect curve from patients with one or two lesions.

"Corroborated Dose" Points in Patients with Multiple Lesions. The data were first analyzed by the "corroborated dose technique." Figures 1 and 2 depict all given tumor doses in 7 of 11 patients with multiple lesions. The corroborated minimum tumor lethal doses are indicated by large dots. These twenty-one corroborated doses were listed in Table 1, and assembled in a scatter diagram (Fig. 5). A free-hand regression curve was drawn for these points and was used as the iso-effect curve. A useful mathematically derived curve could not be obtained because the standard error was too great. Consequently, these data failed to yield a reliable mathematical factor to express the quantitative recovery coefficient. However, the free-hand curve is useful as a clinical guide. This curve will be transferred to Figure 11 for further comparative analysis.

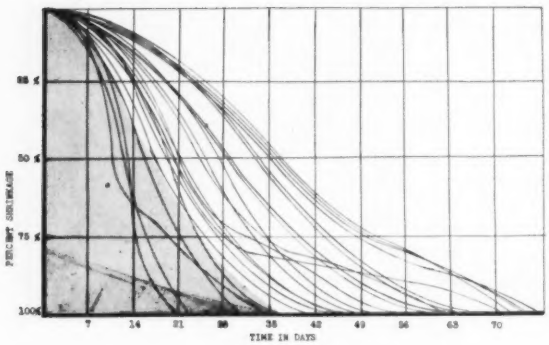

FIG. 6. Shrinkage curves of lesions receiving the corroborated doses illustrated in Figure 5. Seventeen of the twenty-one curves lie within the zone of "slow but complete shrinkage." Thus, minimal lethal doses tend to produce slow but complete shrinkage of a tumor.

It is of interest to study the shrinkage curves produced by the corroborated minimum tumor lethal doses from Figure 5. These curves are assembled in Figure 6. There is a skewed distribution with seventeen of the twenty-one shrinkage curves lying within the zone of "slow but complete shrinkage." Thus, a correlation is observed in that a minimal tumor lethal dose tends to produce slow but complete shrinkage of a tumor.

Analysis of Multiple Lesions on the Basis of Their Shrinkage Curves. The data in patients with multiple lesions were now analyzed according to the type of shrinkage curve. In Figure 7 were assembled all curves of successfully irradiated tumors whose shrinkage was slow but complete, *i.e.*, the curves lay in the unshaded area in the graph. The doses producing these curves were now assembled in the scatter diagram of Figure 8, and a free-hand iso-effect curve drawn. Once again, the standard error was too great to permit mathematical derivation of a recovery coefficient.

The iso-effect curve of "slow but complete shrinkage" (Fig. 8) was compared with that of the "corroborated dose points" (Fig. 5). The similarity of the two iso-effect curves suggests that, in general, the two techniques of analysis of data (*i.e.*, corroborated dose points, and slow but complete shrinkage points) tend to yield similar minimal tumor lethal dose data. The iso-

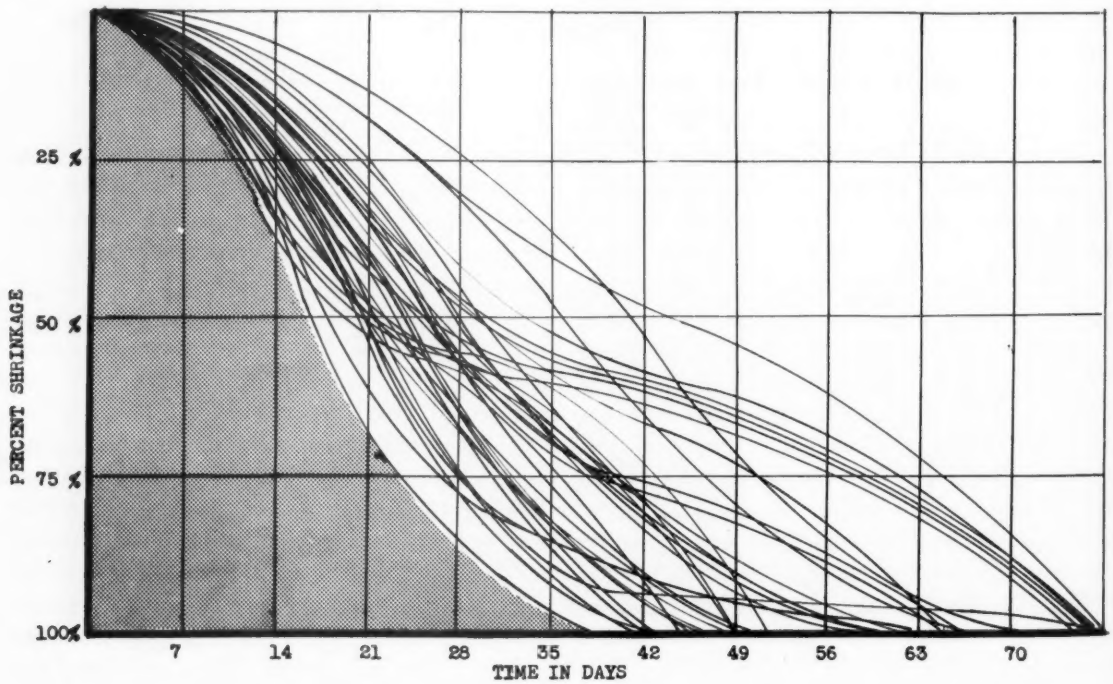

FIG. 7. Curves of successfully irradiated tumors whose shrinkage was "slow but complete." The dosage data from this group of cases are assembled in Figure 8 for special analysis.

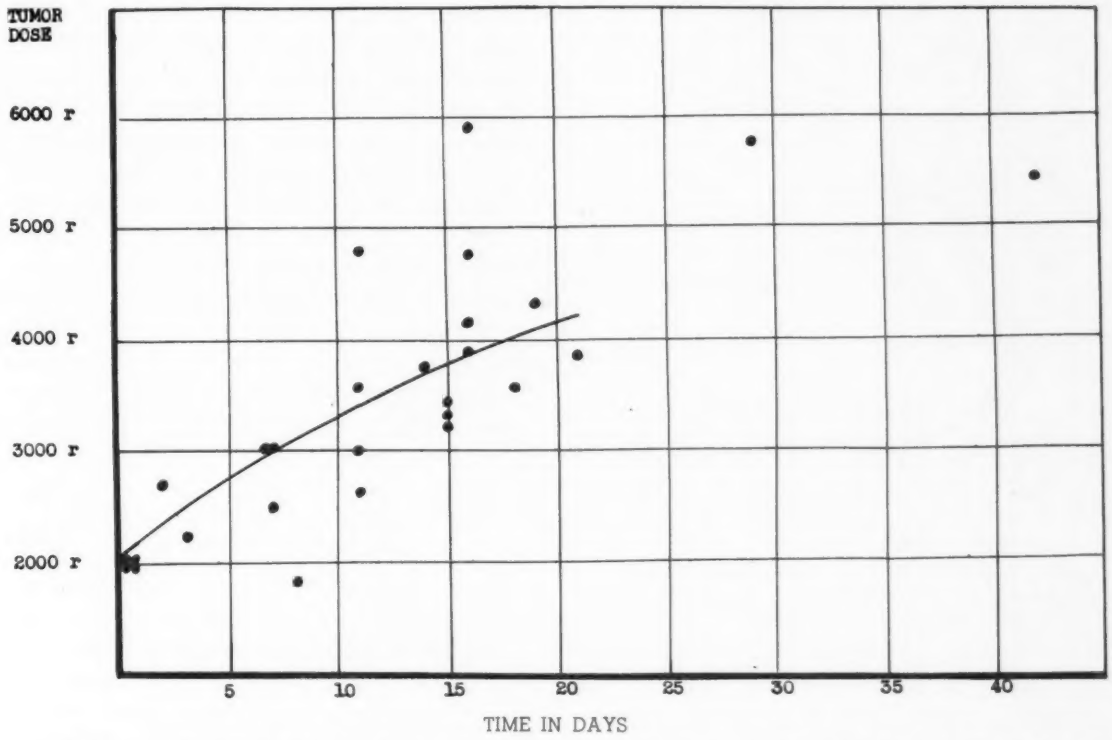

Fig. 8. Scatter diagram of tumor doses which produced "slow but complete shrinkage." The iso-effect curve was drawn freehand. Note the similarity of this iso-effect curve to the one in Figure 5, which was based on "corroborated doses."

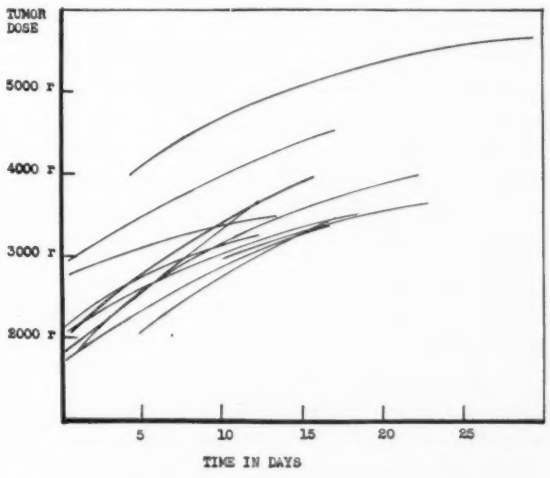

Fig. 9. Iso-effect curves, drawn freehand, of 11 patients with multiple lesions. Some of these curves are illustrated in Figures 1 and 2. A plurality of patients have lesions with similar ranges of tumor lethal dose, but some have lesions which are more radioresistant than the average. The variation in the slope of the curves suggests there may be a different recovery factor for each patient's tumor.

effect curve from Figure 8 will be transferred to Figure 11 for further analysis.

Comment on Iso-effect Curves. These refined techniques for obtaining minimal tumor lethal doses were expected to be superior to previously reported methods for deriving iso-effect curves. Nevertheless, comprehensive mathematical analysis of the data failed to yield a useful recovery coefficient or fractionation factor, because the standard error was too great. It is possible that further refinement of the technique and a much greater number of observations may yield a statistically useful iso-effect curve in spite of the broad range of radiosensitivity of the biologic test object.

The free-hand iso-effect curves for each of the 11 patients with multiple lesions were assembled in Figure 9. Study of this graph indicates that a plurality of patients have lesions with similar ranges of tumor lethal

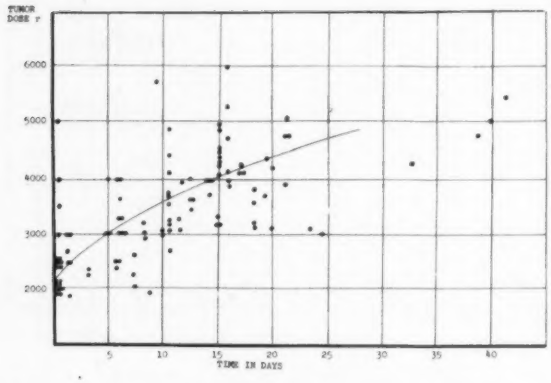

FIG. 10. All successful given doses (106 doses in 29 patients). These include excessive, average and minimum tumor lethal doses. The iso-effect curve delineates higher average dosage than the iso-effect curve based on "corroborated doses" or "slow but complete shrinkage" (Fig. 5 and 8), since it is weighted by excessive doses. It is readily evident why curves based on a single observation in each patient are prone to error.

dose, but some patients have lesions which are more radioresistant than the average. In each case, the biologic recovery process follows a pattern which may result in a useful iso-effect curve for that patient. The variation in the slope of these curves could be due to statistical error, but also suggests that there may be a different recovery factor for each patient's tumor. It is readily evident, from the broad range of tumor lethal dose encompassed by these parameters, that it would be difficult to arrive at a suitable average curve, and that there are one or more factors which are not elucidated by the technique of observation.

Although the above iso-effect curves fail to provide a quantitative recovery factor which is acceptable as a radiobiologic index, nevertheless these curves serve a useful purpose in providing a guide in clinical radiation therapy.

Comparison of Excessive Doses and Inadequate Doses. By comparing these groups of doses, Strandqvist (ref. 6, p. 218) was able to construct iso-effect curves which corroborated his mathematically derived average curve. With few exceptions the excessive doses lay in a region above the average curve, and the inadequate doses below it. Although the placement of his curves was rather arbitrary and their slopes not truly related to the points in his scatter diagrams, Strandqvist found a zone between the excessive and inadequate doses which could harbor his mathematical iso-effect curve.

The same technique was therefore attempted for recurrent breast carcinoma nodules. It failed to yield a useful curve because the range of radiosensitivity for this tumor was so great and the dispersion of the dosage points in the scatter diagram were so erratic that there was no clearly defined zone between excessive and inadequate doses (Fig. 11).

TUMOR LETHAL DOSE FOR RECURRENT BREAST CARCINOMA

There is a wide disparity in the recommended tumor lethal doses for carcinoma of the breast.[1] In the treatment of primary inoperable breast cancer, several authors[1,2,3,5,7] have suggested tumor lethal doses of 6,000 to 8,000 r in 60 to 100 days. This type of fractionation was dictated by empirical clinical considerations. McWhirter irradiated axillary and supraclavicular nodes with a tumor dose of 3,750 r in twenty-one days, following simple mastectomy. His treatment plan, routinely employed in almost all cases, implies that a dose of 3,750 r in twenty-one days is lethal or suppressive to metastatic carcinoma in axillary, supraclavicular and internal mammary nodes. The efficiency of his technique is measured by the five year survival rate. This may not be the proper index by which to judge the effectiveness of a stated dose of radiation in eradicating metastatic breast cancer. The patient may survive with quiescent cancer in the lymph nodes. On the other hand, the patient may have died of remote metastasis, while the regional lymph nodes were sterilized. Uncertainty as to the incidence of lymph node metastasis further beclouds the issue.

The present study was designed in part to obtain additional data on the tumor lethal dose of breast cancer. Variables such as radiation quality, field size, tumor size,

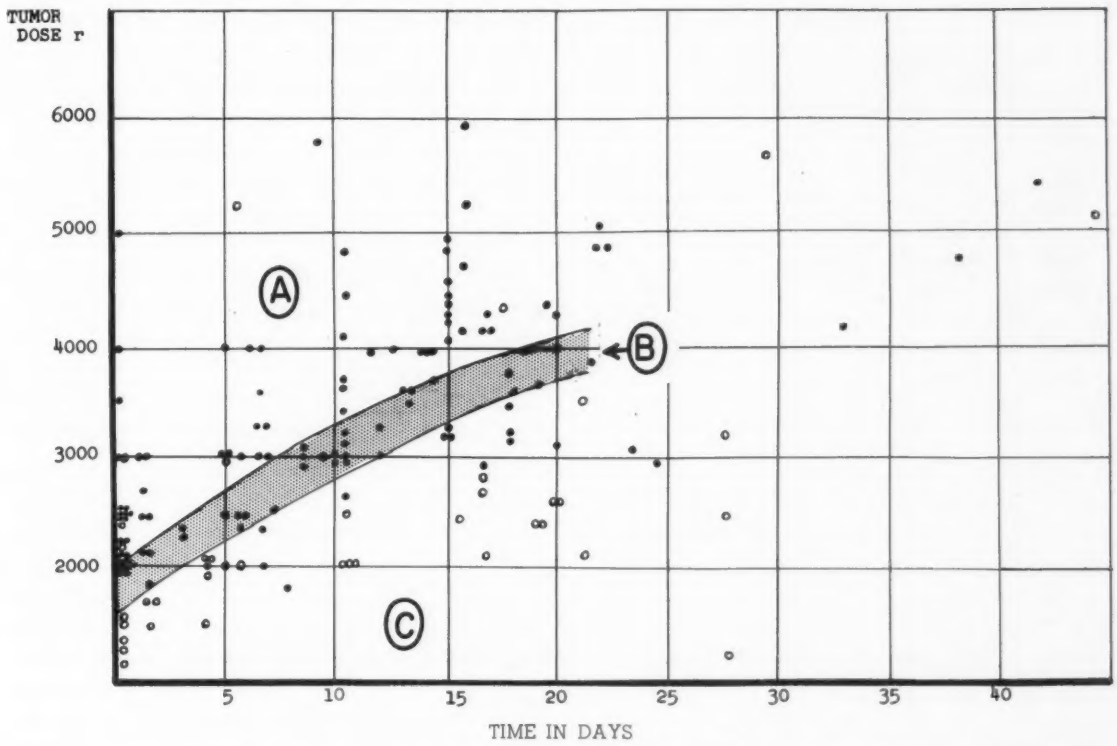

Fig. 11. Summary scatter diagram of all data. The dots are successful doses; the circles are failures. The curve constituting the upper border of the shaded zone is identical to the iso-effect curve in Figures 5 and 8. It constitutes the closest approximation of an iso-effect curve for recurrent breast cancer which could be obtained from this experiment.

The lower parameter of the shaded zone is based on McWhirter's recommended dose of 3,750 r in twenty-one days. Starting with this point a line was drawn parallel to the upper parameter. The shaded zone encompassed by these two curves constitutes an arbitrary dose region for convenience in analyzing data, and divides the graph into three dosage zones. There are 90 per cent successes from the doses in Zone A; 76 per cent in Zone B; and 30 per cent in Zone C

Implicit in these data are two facts: the tumor lethal dosage of recurrent breast cancer has a wide range; and a significant percentage of breast cancer is relatively radioresistant.

and tumor bed were eliminated. It is recognized that a small recurrent chest wall nodule may not have the same response to irradiation as the primary tumor, especially since there is a difference in the tumor bed. Furthermore, a small tumor tends to be more radiosensitive than a large tumor of the same type. Consequently, the tumor lethal dosage for recurrent breast cancer should be smaller than for the primary tumor and metastatic nodes. Comparison with published data[1] indicates that this is not the case. Our tumor lethal dose curve (iso-effect curve) is at a higher level than the crude average curve obtained by Cohen from published data. Allowing for inaccuracies of all data, there remains a certain degree of correspondence in the radiosensitivity of primary, metastatic and recurrent breast cancer.

The following deductions pertain only to recurrent nodules on the chest wall.

The Single Dose for Recurrent Breast Cancer. All the successful given doses in this experiment were assembled in Figure 10 (106 doses in 29 patients). These include excessive, average and minimum tumor lethal doses within the definition of this experiment. Most of the tumor lethal doses given in a single exposure ranged from 1,800 r to 3,000 r, the average being 2,200 r. It is expected that the average figure for

TABLE II

MINIMUM TUMOR LETHAL DOSES FOR VARIOUS OVER-ALL TIMES FOR RECURRENT BREAST CANCER

Days after 1st Treatment	No. of Treatments	Dose (r)	Fractionation Factor (breast)	Strandqvist's Fractionation Factor* (skin)
Single Dose	1	2,000	1.0	1.0
1	2	2,200	1.1	1.26
2	3	2,500	1.25	1.47
3	4	2,650	1.32	1.6
4	5	2,750	1.37	1.7
6	7	3,000	1.5	1.87
9	10	3,300	1.65	2.04
11	12	3,500	1.72	2.13
14	15	3,800	1.9	2.25
19	20	4,200	2.1	2.41
20	21	4,250	2.1	2.43
24	25	4,400	2.2	2.53

* This column is Strandqvist's fractionation factor for skin cancer and is included for purposes of comparison.

these uncorroborated data would be somewhat high. When the corroborated dose data are examined (Fig. 5 and 8), it is seen that for recurrent nodules the average minimum tumor lethal dose given in a single exposure is 2,000 r. Cohen calculated the median curative dose for mammary cancer to be approximately 1,250 r in a single exposure of 200 kv. roentgen rays. This calculated dose is probably too small. Additional evidence concerning the single dose is found in Figure 11. Five lesions, which received a dose less than 1,800 r in a single exposure, recurred. An additional five lesions which received a single dose ranging from 1,800 r to 3,000 r recurred.

The above information can be summarized to provide data of practical use in clinical treatment of recurrent breast cancer. Although irradiation is more efficient when fractionated, nevertheless, when multiple lesions are present, it may be desirable to deliver the tumor lethal dose to each lesion in one treatment. The range of dosage is from 1,800 r to 3,000 r, the average being 2,200 r. It is suggested that the first lesion be given a tumor dose of 2,200 r in one treatment. Its rate of shrinkage and ultimate response will help in determining the dosage for the other lesions.

The Fractionated Dose for Recurrent Breast Cancer. All the observations of this experiment were assembled in Figure 11. The dots represent successful doses as defined by this experiment (*i.e.*, no recurrences of the lesion for at least six months). The circles represent failures. Superimposed on the scatter diagram are two similar iso-effect curves from Figures 5 and 8. These iso-effect curves were derived from "corroborated dose" data and "slow but complete shrinkage data." They were united as a single curve which constitutes the upper margin of the shaded Zone B.

Based on the two similar iso-effect curves, doses producing equivalent effects when delivered over various over-all times are tabulated in Table 11 and compared with Strandqvist's *fractionation factor* for skin cancer. The doses for breast cancer are only approximations as their parent iso-

TABLE III

RELATIVE EFFICIENCY OF VARIOUS OVER-ALL TIME-DOSES FOR RECURRENT BREAST CANCER

Dosage Zones	Number of Lesions Irradiated	Successfully Irradiated		Failures	
		No.	Per Cent	No.	Per Cent
A (Higher than minimal tumor lethal doses)	75	68	90	7	10
B (Dosage in the McWhirter range)	37	28	76	9	24
C (Sublethal dose range)	43	13	30	30	70

effect curves were not mathematically derived.

For convenience in analyzing the data in Figure 11, and because of special interest in the McWhirter technique for treating breast cancer, the scatter diagram (Fig. 11) was divided into three zones: a central shaded Zone B, an upper Zone A, and a lower Zone C. The two similar iso-effect curves from Figures 5 and 8 were merged to constitute the upper parameter of the shaded zone. The lower parameter was based on McWhirter's recommended dose of 3,750 r in twenty-one days. Starting with this point, a line was drawn parallel to the upper parameter. The shaded zone constituted an arbitrary dose region for convenience in analyzing data.

Zone A contained doses higher than the minimum tumor lethal doses derived from this experiment, and also represented doses higher than McWhirter's optimum dosage. Zone B contained McWhirter's optimum dosage, and Zone C contained sublethal doses.

These data are analyzed in Table III.

From Table III and Figure 11, it is evident that the tumor lethal dose for breast cancer occupies a broad range. Some lesions are more radioresistant than others. When irradiating recurrent breast cancer with doses in the McWhirter range (Zone B), there were 24 per cent failures. When larger doses were used (Zone A), there were 10 per cent failures. Therefore, McWhirter's dosage, when applied to recurrent breast cancer, may be inadequate for some of the lesions. Any attempt to apply these deductions to microscopic postoperative residuals and to metastatic axillary lymph nodes is fraught with potentially erroneous deductions. However, based on the above observations and previous experiences with irradiation of primary breast cancer, we are of the opinion that McWhirter's dosage is inadequate for certain cancers. Local tissue intolerance prevents the routine employment of higher dosage.

Since McWhirter's technique achieves survival rates comparable with surgical results despite inadequate dosage in some patients, it is probable that surgery may have inefficiencies of its own.

TABLE IV

CALCULATED RECOVERY FACTOR FOR VARIOUS TISSUES

Author	Tissue	Recovery Factor
Cohen[1]	Breast cancer	0.34
Cohen[1]	Skin (erythema)	0.33
Quimby and MacComb[4]	Skin cancer	0.22
Strandqvist[6]	Skin (erythema)	0.31
Friedman and Pearlman	Recurrent breast cancer	0.25

The Calculated Recovery Factor. A calculated recovery factor has been derived by several authors. It represents the slope of a curve on a log-log graph (see Table IV).

These recovery factors are assembled only for inspection. The data for skin erythema collected by Quimby and MacComb[4] are satisfactory for quantitative statistical treatment. Strandqvist's data[6] for skin cancer are less satisfactory and are useful only for over-all treatment periods of one to fifteen days. The data on breast cancer reported by Cohen[1] does not permit the derivation of a reliable recovery factor. The same is true for the authors' estimated recovery factor.

SUMMARY AND CONCLUSIONS

1. In order to investigate the optimum tumor-lethal dose for recurrent cancer of the breast, and to express this dose in the form of an iso-effect curve or recovery curve, an experiment was designed. Patients with multiple recurrent chest wall nodules were irradiated with different doses delivered in different over-all times. The multiplicity of lesions reduced the errors occurring when a single lesion in one patient was irradiated. Multiple lesions permitted corroboration of the optimum dose for each over-all time period by means of "corroborated dose technique" and "shrinkage curve analysis."

2. Although an iso-effect curve for recurrent breast cancer was obtained by more reliable methods than hitherto described, the standard error of the data was too large to permit the derivation of a mathematical formula for the slope of the curve. The curve was drawn by freehand and consequently the dosage represented by it was approximate.

3. The individual iso-effect curve derived from multiple lesions in a single patient results in a useful iso-effect curve for that patient. However, among different patients there is not only considerable variation in tumor lethal dosage, but also variation in the slope of each iso-effect curve. (The slope of the curve represents the quantitative recovery factor or fractionation factor.) Because of the broad range of tumor lethal dose for breast cancer, it would be difficult to arrive at an average iso-effect curve suitable for all cases.

4. In the practical clinical treatment of patients with multiple recurrent chest wall nodules, a suitable irradiation technique is described, based on a single tumor lethal dose of 2,200 r.

5. The McWhirter technique was reviewed in the light of tumor lethal dosage data derived from this study. His dose of 3,750 r in twenty-one days is probably inadequate for a significant percentage of breast cancers which are radioresistant.

Since McWhirter's irradiation technique achieves survival rates comparable with surgical results it is probable that surgery may have certain inefficiencies of its own.

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DISCUSSION

DR. CHARLES L. MARTIN, Dallas, Texas. The authors are to be congratulated on the type of work which they have undertaken. Much more investigation should be carried out on the influence of the time factor in therapy, and practical information can be obtained only when such studies are made of growing cancer in the human subject rather than in animals.

However, cancer of the breast constitutes a very poor material for such exact determinations because of the extreme variations in its behavior. One patient with this disease may develop extensive metastases and die in a few months, whereas another with a tumor having a similar histopathological structure may live for eighteen years in relative comfort. The authors have partially d'scounted this objection by treating multiple lesions in a single individual but the results so obtained apply to only one type of breast cancer.

Although the disease has been treated for many years with irradiation, very few well documented data have ever been presented to prove that adenocarcinoma of the breast can be destroyed permanently by any type of external irradiation given in safe amounts. Even though Lenz reported a few cases that remained symptom free for seven to nine years after the administration of 6,000 to 8,000 r in one to three months, he described others in which viable tumor could still be demonstrated in areas receiving this same dosage. With these observations in mind, emphasis should be placed on the fact that the doses tabulated on Dr. Friedman's curve are actually optimum regression doses rather than tumor-lethal doses as indicated in the paper. The data which he has supplied will be useful in the palliative treatment of metastases but cannot be expected to produce cures in cancer of the breast.

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INFLUENCE OF THE ELECTROMAGNETIC FIELD UPON THE ACTION OF THE ELECTRON IN THE TREATMENT OF CANCER

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HAVANA, CUBA

WITH the hypothesis that the electron accelerated and concentrated upon the neoplastic field could determine the flocculation of the neogenetic element of cancer, we began a series of experiments irradiating advanced cancer the immediate results of which we will now present.

ACTION OF THE ELECTROMAGNETIC FIELD UPON THE ELECTRON

Magnetic Field. When an electron penetrates in a region where there exists a magnetic field it is subjected to a centripetal force. The intensity of this force depends on the relative position of the initial velocity of the electron in relation to the orientation of the magnetic field. In any case, the value of this force is given by the relation $f = Bvq \text{ sen } \theta$ where:

B = magnetic field intensity (oersteds).
v = initial velocity of the electron (cm. per second).
q = electric charge of the electron (stat-coulomb).
θ = angle formed by the velocity and the magnetic field.
f = resulting force (dinas).

From the above formula we readily see that we may obtain various notable results:

(a) crossed velocity and magnetic field, in this case we have:
$$f = Bvq \text{ sen } 90 = Bvq.$$
(b) parallel velocity and magnetic field, now we have:
$$f = Bvq \text{ sen } 0 = 0.$$
That is, the magnetic field has no action upon the electron.

We know from Newton's second law of movement that $f = ma = mv^2/R$, where a is acceleration (in this particular case angular acceleration) and R is the radius of the circular movement. Nevertheless, by mathematical calculus, which we cannot stop to consider in such a short report, we may derive the following formulas:

$$R = \frac{mv}{aB} \qquad w = \frac{qB}{m} \qquad T = \frac{\pi m}{Bq}$$

w = angular velocity of the rotational movement.
T = period of the rotational movement.

From the above formulas we should note that:

1. The radius of the circular path is inversely proportional to the intensity of the magnetic field.
2. The period of the circular movement is also inversely proportional to the intensity of the magnetic field.
3. The angular velocity is directly proportional to the intensity of the magnetic field.

This movement is characterized by not representing variation of energy to the electron, due to the fact that the path is normal to the force in every instance.

Electric Field. When an electron is in the presence of an electric field it is subjected to a force. This force follows the orientation of the electric field but its direction is contrary to the direction of the electric field. The characteristic of the action of the electric field is to produce an increase in the kinetic energy of the electron given by the formula $W = \frac{1}{2}m\,(v_f^2 - v_0^2)$ where:

m = mass of the electron.
v_f = final velocity.
v_0 = initial velocity.

* Student of the fourth year of Electrical Engineering, Havana University.
Dr. Pedro L. Fariñas died on April 26, 1951. He was not able to complete his experiments and the results obtained up to his death are published as a posthumous homage. We believe that these experiments open a new field in research for the treatment of cancer.

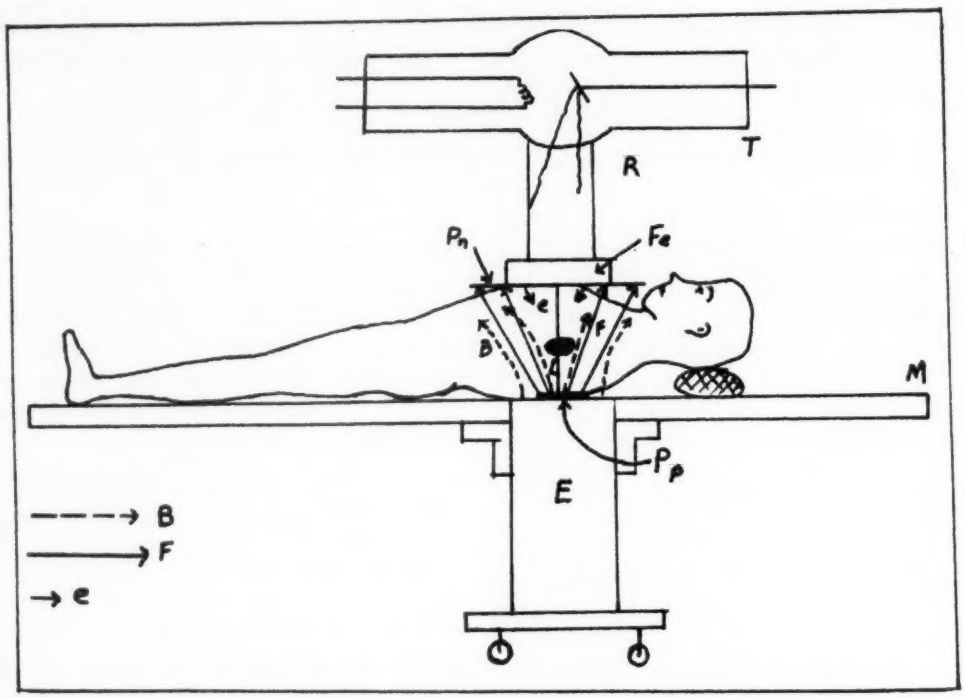

FIG. 1. Shape and disposition of the devices employed in treatments. T=roentgen tube; Fe=heavy metal shield; P=patient; L=lesion; E=electromagnet; Pp=anode of the electric field; Pn=cathode of the electric field; M=special wood table; C=lead cone for complete protection to avoid scattered radiation; R=roentgen ray; e=emerging electrons from the shield of heavy metal; B=magnetic field; F=electric field.

We must consider the relativistic variation of mass with velocity which may be expressed by the Einstein formula:

$$m = \frac{m_0}{\sqrt{1 - \frac{V^2}{C^2}}}$$

m_0=mass when v=0.
m=mass at any instant.
v=velocity of the electron at any instant.
c=velocity of light.

The theory of this action is very delicate and its complete analysis is out of place in this short paper. It is enough that we emphasize that its value lies in the increase of energy that is imparted to the electron by the presence of the electric field.

HEAVY METALS AS SOURCES OF ELECTRONS

It is possible to obtain electrons irradiating heavy metals by means of roentgen rays. In this case the heavy metals act as secondary sources of electrons. Depending on the kilovoltage applied to the roentgen tube we may obtain emission of electrons from the different orbits, that is, from different energy levels. There are three different ways by which this emission may be produced. Let us consider the gamma rays as primary radiation:

1. The gamma photons of low energy level are absorbed by the so-called photoelectric effect. The gamma photons give all their energy to the electrons, part of which is consumed in displacing the electron away from its orbit and the rest is transformed

TABLE I

	Cases	Percentage
Apparently cured	9	20.5
Marked improvement	21	47.7
Little or no improvement	14	31.8
Total cases	44	100.0

TABLE II

RESULTS OBSERVED ACCORDING TO THE LOCALIZATION OF THE TUMOR

	Cases	Per-centage		Cases	Per-centage
Lung			*Mamma*		
Apparently cured	1	6.0	Apparently cured	0	0
Marked improvement	12	70.5	Marked improvement	0	0
Little or no improvement	4	23.5	Little or no improvement	2	100
Total cases	17	100.0	Total cases	2	100
Mediastinum			*Thyroid*		
Apparently cured	0	0	Apparently cured	0	0
Marked improvement	2	50	Marked improvement	0	0
Little or no improvement	2	50	Little or no improvement	1	100
Total cases	4	100	Total cases	1	100
Esophagus			*Liver*		
Apparently cured	1	25	Apparently cured	0	0
Marked improvement	2	50	Marked improvement	0	0
Little or no improvement	1	25	Little or no improvement	1	100
Total cases	4	100	Total cases	1	100
Stomach			*Skull*		
Apparently cured	0	0	Apparently cured	0	0
Marked improvement	1	100	Marked improvement	0	0
Little or no improvement	0	0	Little or no improvement	1	100
Total cases	1	100	Total cases	1	100
Bladder			*Skin*		
Apparently cured	1	33.3	Apparently cured	5	100
Marked improvement	2	66.7	Marked improvement	0	0
Little or no improvement	0	0.0	Little or no improvement	0	0
Total cases	3	100.0	Total cases	5	100
Prostate			*Hodgkin's Disease*		
Apparently cured	1	100	Apparently cured	0	0
Marked improvement	0	0	Marked improvement	2	50
Little or no improvement	0	0	Little or no improvement	2	50
Total cases	1	100	Total cases	4	100

into kinetic energy acquired by the electron. The photons therefore disappear. The energetic relations in this case are given by Einstein's formula $h\nu = \phi + \frac{1}{2}mv^2$

h = Planck's constant.
ν = frequency of radiation.
$h\nu$ = energy of the photon.
ϕ = necessary energy for the displacement of the electron from its orbit.
m = mass of the electron.
v = velocity acquired by the electron.
$\frac{1}{2}mv^2$ = kinetic energy acquired by the electron.

2. When we use gamma radiation of higher energy this phenomenon of emission occurs by Compton's effect.

3. When photons of very high energy collide with the atomic nucleus or reach very close to it, they are absorbed by the atomic nucleus which in turn emits a pair of oppositely charged particles, that is, an electron and a positron.

As roentgen rays are essentially similar to the gamma rays, the above description can also be applied to the roentgen rays, naturally with some limitations. In our method where we have used no higher than

200 kv. the photoelectric effect is 0, the Compton effect is 99 per cent and the production of ion pairs is 1 per cent.

In our experiment we have used iron as the heavy metal (source of electrons) either in solid iron sheet or in fine iron powder, the latter obtained by hydrogen reduction. We have used different thicknesses of iron according to the technique employed.

In all cases we have selected the supine position considering the long duration of the sessions of treatment. The north pole of the electromagnet is placed in the upper part and in direct contact with the patient. The cathode of the electric field is a thin copper sheet of 1 mm. of thickness which at the same time is used to filter the soft electrons. This sheet is 25 by 25 cm.

The anode of the electric field is also a copper sheet 10 by 10 cm. and its thickness is unimportant.

The difference between the dimensions of the anode and the cathode are based on producing a convergent electric field so as to concentrate the electrons upon the neoplastic tissue.

The orientation of the magnetic field is disposed in such a way that it exerts upon the electron a rotational movement around an imaginary axis normal to the position of the patient.

The radius of the circular movement shortens as the electron approaches the anode, on account of the acceleration produced by the electric field. This is the case even though the magnetic field is produced by a D.C. constant electromagnet, its intensity decreases in space as it withdraws from the north pole of the magnetic field on account of the high reluctance formed by the air and the patient.

Biological Action of the Magnetic Field. In regard to studies of the biological action of the magnetic field many experiments have been made beginning in 1890 by Herman and Herrera who reached the conclusion that cellular mitosis is not influenced by the action of the magnetic field. Payne-Scott and Lowe in 1936 pointed out that there exists a special sensibility of the cellular protoplasm due to the presence of the magnetic field.

Later Lenzi found that no difference is observed between the graft of tissue in animals in normal conditions and those modified by previous treatment of the graft by alternating magnetic fields. There exists a notorious delay in the fixing of the graft when the animals, once inoculated, are immediately treated by constant mag-

TABLE III

	Cases	Percentage
Technique 1		
Apparently cured	0	0
Marked improvement	3	75
Little or no improvement	1	25
Total cases	4	100
Technique 2		
Apparently cured	1	5.6
Marked improvement	9	50.0
Little or no improvement	8	44.4
Total cases	18	100.0
Technique 3		
Apparently cured	0	0
Marked improvement	2	100
Little or no improvement	0	0
Total cases	2	100
Technique 4		
Apparently cured	3	50
Marked improvement	0	0
Little or no improvement	3	50
Total cases	6	100
Technique 5		
Apparently cured	0	0
Marked improvement	5	63
Little or no improvement	3	37
Total cases	8	100
Technique 6		
Apparently cured	4	100
Marked improvement	0	0
Little or no improvement	0	0
Total cases	4	100
Technique 7		
Apparently cured	1	50
Marked improvement	1	50
Little or no improvement	0	0
Total cases	2	100

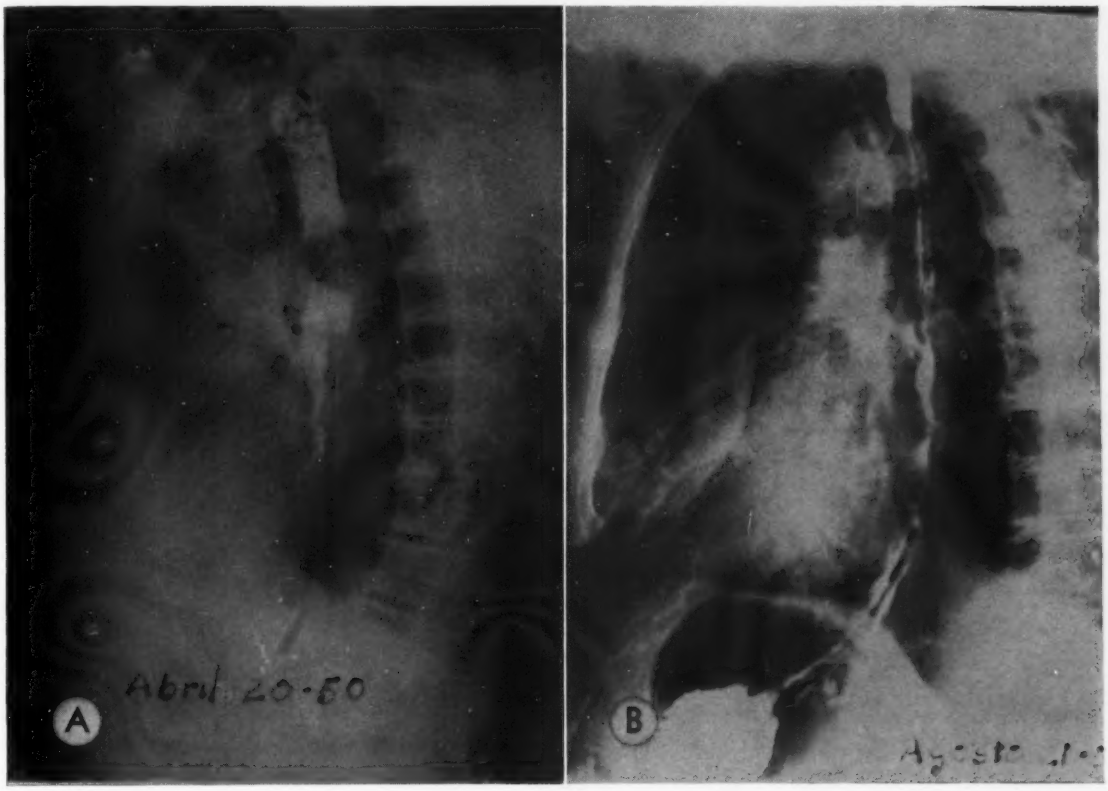

Fig. 2. C. H., aged seventy. Cancer of the esophagus. (*A*) Before treatment. (*B*) After treatment. Technique 2.

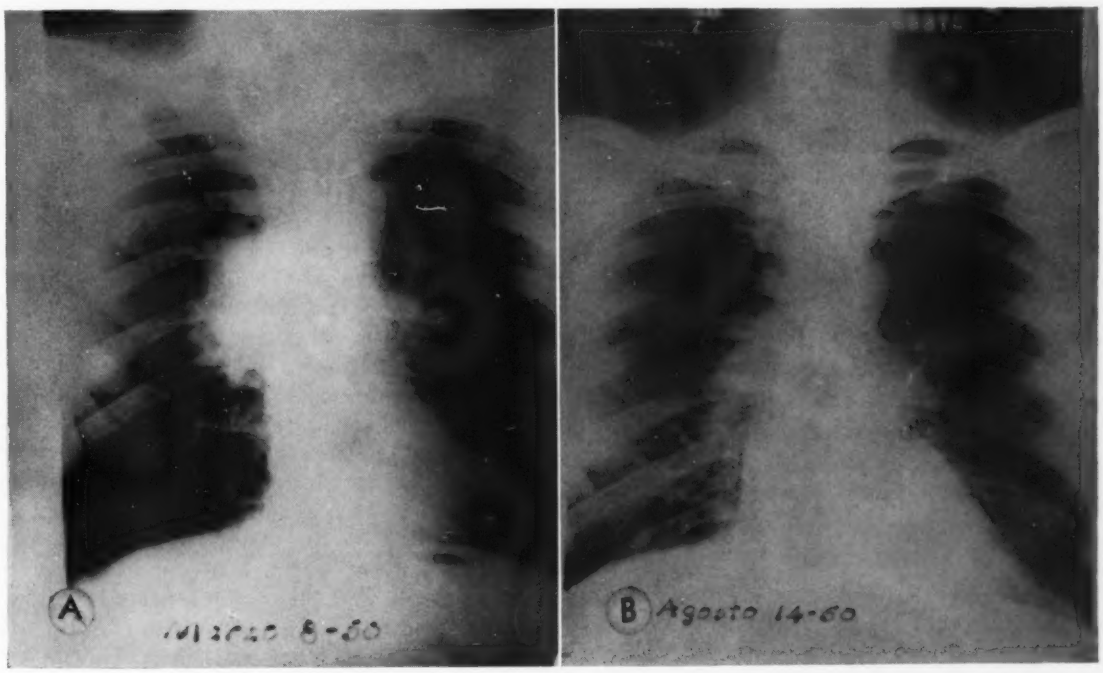

Fig. 3. J. M. A., aged forty-five. Cancer of the lung. (*A*) Before treatment. (*B*) After treatment. Technique 3.

FIG. 4. A. H., aged sixty-five. Hodgkin's disease. (*A*) Before treatment. (*B*) After treatment. Technique 7.

netic fields. With alternating magnetic fields a great reduction in the number of fixed grafts was observed. No variation in the development of the grafts is noted when, once the grafts are fixed, the animals are subjected to magnetic fields.

We wish to point out that never before has the possibility of making neoplastic tissue radiosensitive by the action of a magnetic field been taken into consideration.

If we consider that the immediate action of the radiations upon tissue is to produce an ionization, these ions, positive or negative, must suffer the action of intense magnetic fields thus producing an alteration of the tissue. Also in the case of tissue not exposed to the action of the radiations we can explain the magnetic field action considering the molecular magnetism.

When two atoms differ considerably in their atomic number and they join each other, the resultant molecula is not symmetrical and the pole of positive charge does not coincide with the pole of negative charge, thus from this bipolarity a small magnet is formed. With intense magnetic fields this small magnet would be submitted to their action, obtaining therefore a modification of tissue.

Techniques.

1. Voltage of the roentgen tube 200 kv., filament current 5 ma. Protection of the neoplastic tissue by 1 inch of solid iron. Dosage: 0.25 r per minute and 2.5 r per hour during the first five sessions of 1 hour duration each, divided in ½ hour anterior field and ½ hour posterior field; the other five remaining sessions, with a protection of 2 inches of solid iron over the lesion and ¼ inch of solid iron on the surrounding field. No roentgen rays reach the lesion directly. The surrounding fields are given 1.87 r per minute and 2.5 r per hour. A total of 10 sessions in 10 continuous days.

2. Voltage of the roentgen tube 160 kv., filament current 5 ma. Protection of the neoplastic tissue and surrounding fields by ½ inch of solid iron. Dosage: 0.93 r/min. and 2.5 r/hr. A total of 10 sessions in 10 continuous days. Each session of 1 hour.

3. Voltage of the roentgen tube 160 kv., fila-

ment current 5 ma. Protection of the neoplastic tissue and surrounding fields by $\frac{1}{4}$ inch of solid iron. Dosage: 1.8 r/min. and 2.5 r/hr. A total of 10 sessions in 10 continuous days; $\frac{1}{2}$ hour sessions.

4. Voltage of the roentgen tube 160 kv., filament current 5 ma. Protection of the neoplastic tissue and surrounding fields by 1 inch of solid iron. Dosage: 0.15 r/min. and 1.25 r/hr. A total of 10 sessions in 10 continuous days; 1 hour sessions.

5. Voltage of the roentgen tube 86 kv., filament current 5 ma. Protection of the neoplastic tissue by 2 inches of iron powder. Dosage: 0.00 r/min. and 2.5 r/hr. A total of 5 sessions in 5 continuous days; 1 hour sessions.

We also used this technique giving the treatment for 5 hours continuously in 1 day.

6. Voltage of the roentgen tube 200 kv., filament current 5 ma. This technique was used in cancer of the skin in which the lesion was covered by a suspension of colloidal iron. Dosage: 25 r/min. and 2.5 r/hr. A total of 3 sessions in 3 continuous days; 12 minute sessions. This treatment is repeated one week later.

7. Voltage of the roentgen tube 160 kv., filament current 10 ma. An amount of iron powder contained in a receptacle of aluminum the dimensions of which are 20 by 20 cm. and the height of which is 2 inches is irradiated during 1 hour. This receptacle is carried to the room where the patient is ready for treatment and he is submitted to the secondary radiations emanating from the irradiated iron. This treatment lasts 1 hour. After this hour has been completed the iron is again re-excited for 15 minutes after which the patient is again treated for 1 more hour. This process is continued until 5 hours of treatment are completed in one day.

This treatment is repeated a week later.

Results Immediately Obtained. In our cases we have been able to see that among the varied symptoms which the patients had (according to the localization of the tumor) pain has been the first symptom to disappear; in some cases, pain disappears within forty-eight hours after the first treatment has been applied. This notable alleviation of pain or even its total disappearance in a short time is observed, we repeat, whatever the localization of the tumor may be; thus, the psychological condition of the patient improves notably, reaching in some cases a euphoric state.

The other symptoms improve more slowly, disappearing a few days after the ending of treatment in most of the cases.

Statistics. We have classified our cases according to obtained results as follows: apparently cured, marked improvement and little or no improvement.

Apparently Cured: We have classified in this group all those cases in which the total destruction of the tumor and total remission of symptomatology was proved. We say "apparently cured" because we understand that sufficient time has not elapsed to assume the absolute cure of the patients.

Marked Improvement: In this group are classified all those cases in which, even though there was a total remission of symptoms, the complete destruction of the tumor was not proved.

Little or No Improvement: In this group are classified all the cases in which the symptoms were improved little or not at all and the tumor was enlarged or of a similar character to that observed at the beginning of the treatment.

SUMMARY

The influence of the magnetic field and accelerated and concentrated electrons in the treatment of various types of advanced cancer is reviewed.

Statistical tables are presented in regard to the results obtained.

Dra. Laura Fariñas
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A CLINICAL INVESTIGATION OF THE CHRONIC EFFECTS OF RADIUM SALTS ADMINISTERED THERAPEUTICALLY (1915–1931)*

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HISTORICAL

WITHIN two decades of the isolation and identification of radium by the Curies, the therapeutic administration of this element, its isotopes, and other closely related radioactive elements had become an established medical procedure. As early as 1914, an extensive literature had developed relating to the therapeutic efficacy of radon administered by inhalation, orally, and parenterally; of soluble radium salts, orally and parenterally; of thoron by inhalation; and thorium X orally and parenterally. Mesothorium, radiothorium, and polonium had also been employed.[8–11,28,35,40,48,58] The diseases for which these radioactive materials had been used were legion and included such clinical diversities as syphilis, hypertension, gout, infectious polyarthritis, "muscular rheumatism," leukemia, pernicious and other anemias, epilepsy, and multiple sclerosis.[1,2,5,6,12,13,15–20,29,30–32,34,36,37,46,47,49,51–53,56,57,62]

By 1913, the parenteral and oral use of radium and its congeners was well established in the United States and Europe. Rowntree and Baetjer[58] summarized the treatment of 1,038 persons injected with radium in various clinics and reported clinical improvement in 837. Early enthusiasm was based on an apparently high degree of therapeutic efficacy, the absence of clinically observable acute toxic manifestations, and the erroneous belief that the radioactivity was totally eliminated from the body after a period of months. The appearance of toxic effects at a very late period (ten to twenty years after employment of the medication) was not considered. Parenteral dosages, administered weekly, varied from 5 to 100 μg. and were continued to a total dose as great as 1,000 μg. On occasion, 1,000 μg. of radium were administered as a single dose. The scientific literature that appeared between 1913 and 1916 contains case reports of more than 70 individuals treated intravenously with between 50 and 1,000 μg. of radium.[11,12,13,50,51,53,54]

Until the late 1920s, the administration of radium, orally and parenterally, and radon and its daughters, orally, parenterally, and by inhalation, flourished. One physician[25] stated in 1921 that, since 1913, he had given over 7,000 injections of radium element in doses ranging from 10 to 100 μg.[21–24] Over the years, he had treated "several thousand" patients with intravenous injections of radium.[26] Data published by other clinicians record the administration of radium to at least 100 patients, as late as 1926–1927. It is also known that the administration and taking of soluble radium salts has continued sporadically to the present time in the United States. There must, therefore, exist in the United States a residuum of perhaps hundreds or thousands of individuals who have radium salts deposited in their bodies.

With the publication, in 1924, of the first paper by Blum,[14] soon followed by that of Hoffman,[33] and the classic papers of Martland and his co-workers,[41–45,55] evidence became available of the long-term retention in the body of radium, mesothorium, and radiothorium with the attendant delayed toxic effects manifested as bone necrosis, neoplasia, and hematopoietic dyscrasias.

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However, the therapeutic use of radium salts administered orally and parenterally continued until 1932, at which time radium for internal administration was removed from listing in the New and Nonofficial Remedies of the American Medical Association.

Data derived from experiments done on small laboratory animals with the alpha particle-emitting, long-lived elements are not readily extrapolated to man because of marked differences in life span, in geometry of the biologic systems studied, in metabolism, and in the rates of calcium deposition and turnover. Fundamental in the calculations involved in the determination of permissible body burdens of radioelements deposited in bone have been the studies done on the radium dial painter cases by Martland *et al.* Inherent, however, in an unbiased analysis of these data is the requirement that the groups studied not be unduly weighted with individuals seen by physicians *first* because of the appearance of clinical symptoms. It seemed desirable to the authors to extend the past and current investigations by Aub, Evans, Hemplemann and Martland,[4] by an analysis of data derived from as many patients as were available from the residuum that had received radium therapeutically and that had been selected, if possible, for this reason alone, rather than on the basis of their seeking out medical care because of the presence of symptoms. Close attention has been directed to an evaluation and detection roentgenographically of a "minimal" bone lesion that would objectively indicate bone damage not necessarily associated with clinical symptoms. To this end, we undertook to find as many individuals as possible in the Chicago area who had been given radium therapeutically, as well as those included in the original Elgin State Hospital series.

CLINICAL MATERIAL

During 1930 through 1932, 39 psychotic patients who were hospitalized at the Elgin State Hospital were injected intravenously at weekly intervals with a soluble salt of radium in doses of 10 μg. of the element. The total dosages ranged from 70 to 450 μg. Most of these patients were schizophrenic and the majority were in the twenty to thirty year age group at the time of treatment. Careful physical determinations of total body gamma radiation and exhaled radon were done on the group and reported in the literature.[7,59–61] Radon and gamma-ray measurements were done at from one to six months and again at about one year following completion of the therapeutic course.

Through the cooperation of the United States Radium Corporation and the staff of the Elgin State Hospital, we were able to find the original data sheets of Schlundt *et al.*,[59] which included the original list of patients treated at the hospital. Of the original group of 39 patients, 19 have been located in the state hospitals of Illinois and have been studied. Preliminary studies[38] of these patients have been reported. The status of the entire Elgin group has been analyzed (Table 1), and it appears that none of the individuals who died had significant clinical changes related to the administration of radium. It has been pos-

TABLE I

THIRTY-NINE PATIENTS KNOWN TO HAVE HAD RADIUM CHLORIDE INTRAVENOUSLY AT THE ELGIN STATE HOSPITAL 1930–1931

I. Status of 28 of the 39 Patients
  19 patients have been located and evaluated
    12 still remain at Elgin
      4 were transferred to the Manteno State Hospital
      3 have been discharged
    3 other patients have been located but not evaluated
  6 patients' status have not been determined
II. Cause of Death in 11 Patients Who Have Died
  4 pulmonary tuberculosis
  2 hypertensive heart disease
  1 suicide
  1 intestinal obstruction
  1 kidney disease
  1 bronchopneumonia
  1 carcinoma of stomach

TABLE II

ESTIMATED BODY BURDEN OF 20 INDIVIDUALS ADMINISTERED RADIUM THERAPEUTICALLY IN THE CHICAGO AREA 20 TO 30 YEARS AGO

Patient Code	Sex	Dose Administered (μg).	Age at Examination	Approximate Age in Years at Time of Administration	Route of Administration	Estimated Body Burden in μg. of Radium			Radon/Radium Ratio
						γ-rays	Rn	Total	
119	M	Unknown	30	6–8	Orally	0.01	0.02	0.03	0.66
115	F	Unknown	72	59	Intramuscularly			0.06	
109	F	Unknown	65	33	Orally	0.03	0.04	0.06	0.67
120	M	Unknown	32	8–10	Orally		0.097		
117	M	Unknown	44	16	Intramuscularly	0.11	0.4	0.5	0.80
101[1]	F	250	73	47	180 μg. orally; 70 μg. intramuscularly	0.1	0.41	0.5	0.80
116	F	160	53	31	Intravenously	0.21	0.5	0.7	0.71
105	F	Unknown	74†	Unknown	Unknown	0.17	0.69	0.86	0.80
113	M	Unknown	58	30	Intramuscularly	0.4	0.6	1.0	0.60
112	F	Unknown	48†	20	Orally	0.7	0.6	1.3	0.46
102	M	Unknown	81	Unknown	Unknown	0.5	1.1	1.6	0.69
114	F	Unknown	57	Unknown	Unknown	0.61	1.0	1.6	0.62
111	F	300–400	67	33–36	Intramuscularly	2.0	2.2	4.2	0.52
110	M	700–800	82	51–58	Intramuscularly	1.6	3.8	5.4	0.42
106	M	Circa 200	54	25	Intramuscularly	2.3	3.3	5.6	0.59
103	F	Unknown	63	Unknown	Unknown	2.27	4.30	6.57	0.65
K.B.	F	Unknown	66	46	Orally			2–10*	
104[2]	M	Circa 330	78†	51–56	Intravenously	5.0	5.7	10.7	0.54
108	F	Unknown	69	37–42	Unknown	4.4	6.9	11.3	0.61
107	M	More than 250	70	43	Intramuscularly	4.4	9.5	13.9	0.68

[1] Patient III.
[2] Patient VII—as described by Barker and Schlundt.[7]
* Estimated in 1947 from count of γ-rays by Geiger-Müller counter.
† Age at death.

sible to find and study an additional 20 individuals in the Chicago area who were given radium as a therapeutic procedure from twenty to thirty years ago (Table II). In addition, 6 "dial painters" from an Illinois watch dial factory have been studied.

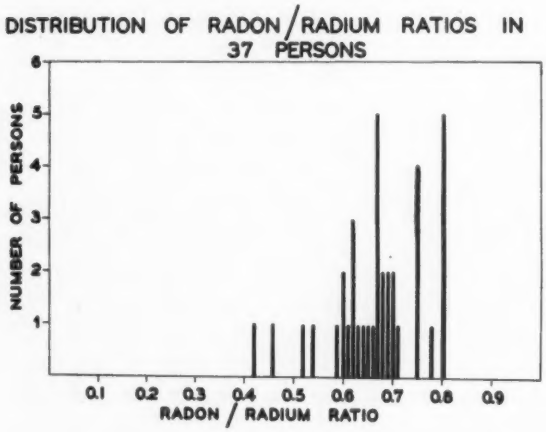

FIG. 1

In this study, we have fulfilled the following criteria: a period sufficient for the development of "late" effects has elapsed (20 to 30 years); the patients were selected (with but 6 exceptions) on the basis of having received radium and not because they were ill. Conclusive evidence has been secured by the authors from the papers furnished by the United States Radium Corporation that the luminescent paints used by the young women studied in the Martland-Aub series were composed of the phosphor and continually varying propor-

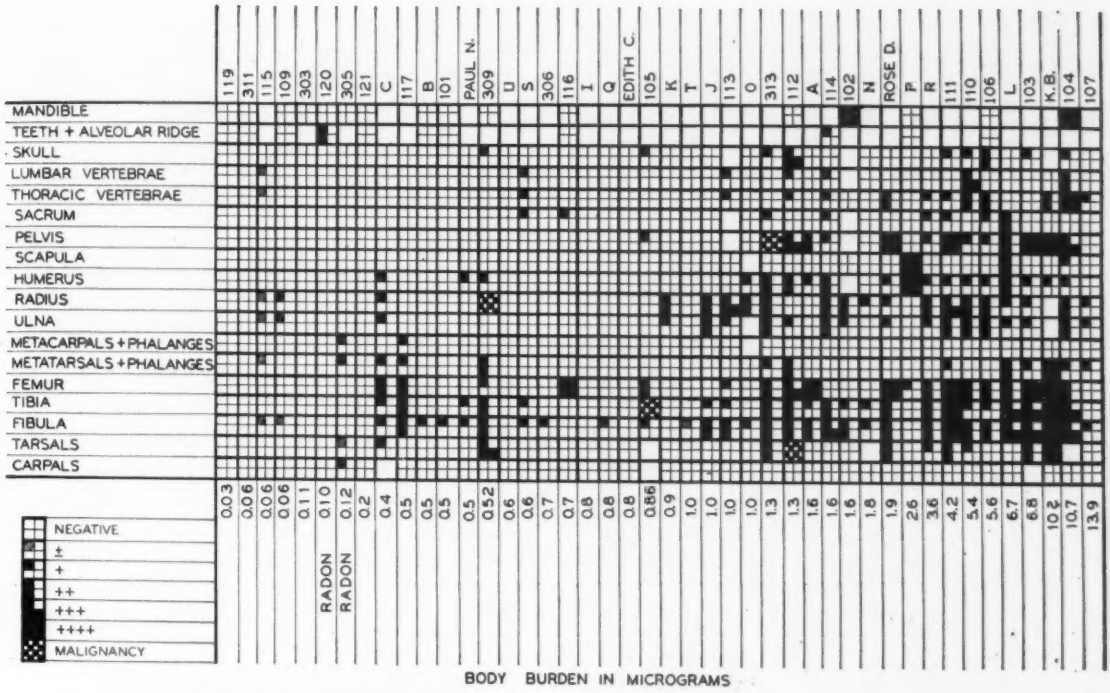

Fig. 2. Chart showing the relationship between body burden of radium and frequency and severity of bone lesions.

tions of radium and mesothorium, mesothorium and radiothorium, radium and radiothorium, or radium or radiothorium alone. It thus seems impossible at present to quantitate relative effects of these related radioelements in any one individual of the Orange, New Jersey, series.

Total retained radium and radon/radium ratios were determined in all patients (Fig. 1) according to the methods described in papers one[40a] and two [61a] of this series.

CLINICAL STUDIES

Roentgenographic examination of the entire skeleton was done on all patients. In addition, as controls, roentgenograms were made of the extremities of 40 persons selected at random while undergoing a general physical examination at the Health Division of the Argonne National Laboratory. Each roentgenogram was examined, and the result of the study plotted on a master chart (Fig. 2). Bone lesions were expressed as being questionably present, or of 1+, 2+, 3+, or 4+ severity.* All hematologic studies (Table III) were carried out by one of the authors (E. S.); the general examinations were made by the physicians of the group.

ROENTGENOGRAPHIC FINDINGS

The major bone changes occurring in persons with radium deposited in the skeleton have been described in detail by Martland,[42,43,45] and Aub *et al.*[4] In this evaluation of the late effects of radium, an attempt was made to recognize and describe the bone lesion, minimal in extent and appearing earliest, that could be associated with the presence of radium in the skeleton. It was soon recognized that since the pathogenesis of the bone changes would not be singular, so also there could not be only one specific bone lesion.

The "major" roentgenographic changes

* Those lesions classified as 1+ fall in Group I (*vide infra*), as 2+ fall in Group II, and as 3+ and 4+ in Group III.

TABLE III

HEMATOLOGICAL STUDIES*

14 Patients at Elgin State Hospital Who Had $RaCl_2$ Intravenously—1930–1931
10 Patients in the Chicago Area Who Had $RaCl_2$ Intravenously, Intramuscularly, or Orally—1920–1930†

Patient Code	Estimated Total Body Radium	Date	Erythrocyte Count (per cu. mm.)	Hemoglobin (photoelectric method) (g./100 cc.)	Leukocyte Count (per cu. mm.)	Polymorphonuclear Count (%)	Stabs (%)	Metas (seen in 300 cells)	Lymphocytes (%)	Monocytes (%)	Eosinophils (%)	Basophils (%)	Condition of Red Cells on Differential	Mitotic Lymphocytes (total seen)	Bilobed Lymphocytes (total seen)	Immature Lymphocytes (total seen)	Slightly Immature Lymphocytes	Atypical Lymphocytes	Degenerating Cells	Cells with Vacuoles	Giant Platelets	Atypical Platelets	Sedimentation Rate (mm. in 1 hr.)	Hematocrit (%)
109	0.07	1/31/51	467	13.7	5,650	48	—	—	40	7	5	—	Normal	—	—	—	—	Occ.		—	Occ.	—	35.0	41
		4/27/51	424	12.6		49	—		47	2	1	1	Normal			1	—	—		—	—	—	—	—
Paul W.	0.4	8/29/50	491	14.5	25,800	84	2	1	10	3	0	0	Normal	1	—	1	—	—	1 lymph	—	—	—	30.0	47
		5/10/51	485	13.0	12,600	63	—	—	32	2	2	1	Normal	—	—	1	1	—	—	1 eosinophil	—	—	24.0	45
B.	0.4	5/10/51	487	16.0	11,000	68	—	—	26	4	1	1	Normal	—	—	—	Few	—	—	2 monos	—	—	6.0	46
117	0.5	3/16/51	440	13.2	7,700	53	2	—	34	4	6	1	Slight anisocytosis	2	—	1	3	—	—	1% eosinophil	Few	Few	22.0	44
U.	0.7	5/ 9/51	448	11.7	9,500	88	1	—	32	4	2	2	Normal	—	—	—	2	—	—	1 lymph	—	—	42.0	41
I.	0.9	5/10/51	406	12.5	8,600	67	1	—	25	2	4	1	Normal	—	—	—	—	—	—	2 eosinophils	—	—	47.0	37
Q.	0.9	8/29/50	497	16.5	17,000	88	1	—	9	2	—	—	Normal	—	—	—	4	—	polys sl toxic	—	—	—	35.0	47
		5/10/51	515	17.0	9,600	70	2	1	24	3	1	—	Normal	—	—	1	2	—	2 lymphs	—	—	—	20.0	47
116	1.0	11/17/50	467	12.7	11,200	72	—	—	26	1	1	—	Normal	—	—	—	—	—	—	2 lymphs	—	—	27.0	39
K	1.0	5/ 9/51	431	12.2	9,500	52	1	—	44	1	2	—	Slight anisocytosis Few stippled red blood cells	—	1	—	3	2 (transitionals)		—	—	—	42.0	41
O	1.1	5/ 9/51	435	12.5	8,100	53	1	—	33	2	10	1	Normal	—	—	1	2	—	—	2 eosinophils	Few	—	27.0	41
J.	1.2	8/17/50	467	13.6	11,800	55	—	—	42	2	2	—	Normal	—	—	—	1	—	—	—	—	—	42.0	44
		5/ 7/51	445	13.6	7,200	75	1	—	23	1	—	—	Normal	—	—	—	3	—	—	1 lymph	—	Few	29.0	42
113	1.2	4/25/51	485	15.2	7,700	51	3	1	44	1	1	—	Normal	3 lymphs with lg. azure gran.			2	—	—	—	—	—	12.0	48.5
T.	1.2	8/22/50	472	14.5	12,300	57	—	—	21	3	18	1	Normal	—	—	—	—	—	—	—	—	—	40.0	46
		5/ 7/51	420	13.0	8,400	52	2	—	25	6	15	—	Normal	—	—	—	1	—	—	4 eosinophils 1 poly.	—	—	48.0	41
102	1.5	4/ 4/51	504	14.9	10,000	61	3	1 basophilic	30	4	1	1	Normal	1	3	—	3	—	—	3 lymphs	Few	Few	13.0	52
114	1.7	4/27/51	426	13.8	6,400	64	—	—	34	1	1	—	Normal	—	—	—	—	—	—	—	—	—	32.0	43
112	1.9	1/11/51	374	11.2	4,900	59	2	—	36	1	2	—	Marked anisocytosis Mod. poikilocytosis Slight hypochromia	Polys toxic			—	Few		1 lymph with vacuoles	—	—	54.0	36
		5/ 1/51	414	11.5	7,550	63	2	—	30	2	3	—	Slight anisocytosis and hypochromia	Polys toxic			3	—	2 lymphs					
A.	2.2	8/17/50	450	12.2	6,700	71	—	—	25	4	—	—	Slight anisocytosis and hypochromia	—	—	—	Few	—	—	—	—	—	32.0	41
		5/ 7/51	493	12.2	10,300	65	—	—	32	1	2	—	Slight anisocytosis and hypochromia	—	1	—	1	—	—	—	—	—	38.0	42
N.	2.4	8/17/50	435	13.2	8,900	58	—	—	38	2	2	—	Slight anisocytosis and hypochromia	—	—	—	Few	—	1 lymph	—	—	—	40.0	42
		5/ 7/51	435	13.5	6,600	51	—	—	43	2	2	2	Slight anisocytosis	1	—	—	1	—	—	—	—	—	48.0	40
P.	2.8	5/10/51	440	12.9	9,400	69	1	1	26	3	1	—	Normal	—	—	3	Few	—	—	1 lymph	—	—	43.0	41
R.	4.1	8/22/50	424	11.5	6,150	55	1	—	46	4	3	1	Slight anisocytosis	—	—	—	2	—	—	—	—	—	47.0	40
		5/ 7/51	430	12.2	8,200	47	—	—	49	2	1	1	and hypochromia	1 binucleated poly. Several lymphs with large azure gran.					Few polys	—	Few	—	41.0	39
106	6.1	4/17/51	494	16.0	9,750	65	1	—	31	1	2	—	1 Polychromatic red cell	—	—	—	—	Occ.	—	1 poly 1 eosinophil	—	—	20.5	50.5
L.	7.2	5/ 9/51	382	11.5	7,600	64	1	—	25	2	8	—	Moderate anisocytosis and hypochromia	—	—	—	—	—	—	1 mono 1 eosinophil	—	—	37.0	38
104	10.2	3/13/51	356	11.9	10,600	67	7	1	23	1	1	1	Moderate anisocytosis Slight poikilocytosis and hypochromia	Few toxic polys			4	—	—	Few polys and eosinophils occ. lymph	—	—	38.0	35
108	12.3	5/10/51	485	14.0	7,000	68	—	—	27	4	1	—	Normal	—	—	1	2	—	2 lymphs	2 lymphs	—	—	32.0	44

* Hematological studies made by Ellen Skirmont.
† Chicago patients.

seen in the Martland and Aub series of patients, mostly from the radium dial industry, have been well described by these observers. The following excerpts from an early article by Martland[42] give the essential features of bone damage in the "dial painter" cases.

As the jaw necrosis was the outstanding feature of the early cases, our attention was fixed mainly on this symptom and little attention was paid to changes in the other bones of the skeleton, although an extensive coxa vara was noted in one case. . . . These bones were thought to contain a greater amount of radioactivity than did other bones of the skeleton. It was not until I began to see the later cases that I realized that this was not the correct interpretation of the lesion. In the later cases, localized pain had occurred in other bones and destructive lesions were demonstrated by the roentgenograms. These lesions were particularly noticeable in bones exposed to trauma, like the scaphoid in the feet (tight shoe), and those carrying considerable weight, such as the spine and heads of the femurs. . . . In other words, the lesions were quite evenly distributed over the entire skeleton, although in each individual bone the distribution, while diffuse, was distinctly patchy and concentrated in areas.

Fig. 3. Small and large isolated areas of bone resorption in the radius and ulna.

Fig. 4. Areas of bone resorption in the humerus of a patient carrying a body burden of 6.7 μg. of radium.

Martland,[43] as early as 1931, demonstrated the roentgenographic appearance of the skull showing areas of rarefaction, and Flinn,[27] in 1934, considered these "punched out" areas in the skull to occur with sufficient frequency in "radium dial" patients to assume diagnostic value. Aub[3] in 1938, described the roentgenographic changes in

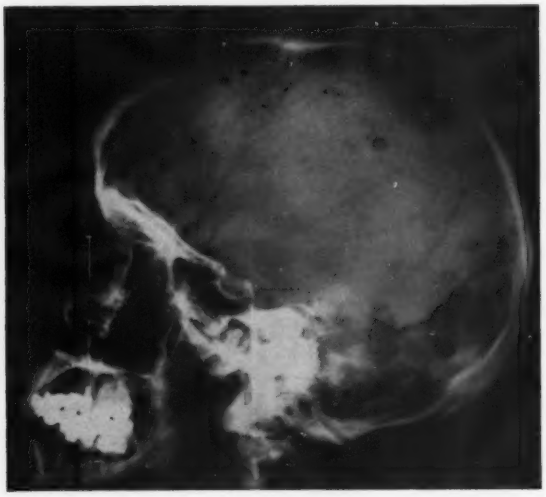

FIG. 5. Multiple "punched out" areas of bone destruction in the skull, in patient with a body burden of 1.3 μg. of radium.

the heads of the humeri and femora as well as in the tarsal bones.

ROENTGENOGRAPHIC CLASSIFICATION OF PATHOLOGIC CHANGES

Group I. The *significant* minimal roentgenographic lesions observed by the authors have been multiple 1 to 2 mm. by 5 to 20 mm. areas of decreased density occurring with great frequency and located most prominently in the fibula, tibia, radius, ulna, humerus, and femur (Fig. 3 and 4). Related lesions are the "punched out" areas (Fig. 5) of decreased density in the skull that closely resemble, but must be differentiated from the lesions of multiple myeloma. The skull lesions vary in size from 2 to 10 mm. in diameter and may be irregular or smooth in outline. In one patient (Patient 112), we observed a striking increase in the number and size of these skull lesions over a three year period.

When the small areas of decreased density that occur in the long bones become multiple, they lend a "streaked" appearance to the bone (Fig. 6). Infrequently, these lesions may be solitary and as large as 3 to 4 cm. in diameter. In these instances, the site of predilection is usually the tibia or femur. Occasionally, these larger lesions may be accompanied by diffuse "streaking" of the bone.

We have noted small areas of bone resorption in the humerus, radius, ulna, phalanges, femur, tibia, fibula, and trasals in a patient carrying a body burden of 0.4 μg. of radium. It is concluded that this lesion is *not diagnostic of*, but is *most suggestive of* the presence of significant quantities of radium in the skeleton, especially when noted in the cortex of the extremities of the long bones.

That these areas of decreased density do not represent roentgenographic artefacts but are true areas of bone resorption has been demonstrated by the histologic and autoradiographic studies of thin bone sections by Looney and Woodruff[39] (Fig. 7). Accordingly, we believe this to be the mini-

FIG. 6. Multiple small areas of bone destruction and resorption lend a "streaked" appearance to the long bones.

mal roentgenographically recognizable lesion. Its importance lies in the fact that it can be noted in patients who are *asymptomatic*, in the absence of other more marked lesions, and it occurs with a lesser body burden than any other lesion.

Group II. Areas of apparently increased density usually associated closely with areas of decreased density and showing varying degrees of change in trabecular pattern are characteristic of this group (Fig. 8).

Certain bones have areas of apparently increased density of irregular and inconstant shape and size. The lesions may be as large as 1 centimeter in diameter, resembling the localized bone infarcts seen in "caisson disease," and are not specific for radium poisoning. They appear most frequently in the alae of the ilium and the heads of the femur and humerus.

Fig. 7. Photomicrograph of localized areas of bone destruction in tibial cortical bone from patient with total body burden of 1.3 μg.

Fig. 8. Extensive changes in the trabecular pattern in the radius and ulna.

A variant of this lesion is characterized by multiple areas of bone resorption contiguous with areas of apparently increased bone density and a broadening and coarsening of the trabeculae, which lend a "mottled" appearance to bone (Fig. 9). This

FIG. 9. Scattered areas of apparently increased density in the alae of the ilia with coarsening of the normal lacy trabeuclar pattern in patient carrying body burden of 1.3 μg. of radium.

is seen in the ilium, the shafts of the femur and humerus and their heads (Fig. 10), and in the glenoid process of the scapula where it may be so extensive that the bone appears "moth eaten."

The bodies of the vertebrae in several instances show a marked coarsening of the structure of the trabeculae of the spongiosa. In a few instances, small areas of apparently increased density have been noted in the vertebral bodies. It is believed that although these changes are *not* diagnostic of radium poisoning, and may be nonspecific reactions to several noxious processes, they do occur with increased frequency in patients retaining large burdens of radium (Fig. 11, 12 and 13).

Group III. Certain "major" changes have also been observed in the bones of the patients in our series. From the roentgenographic standpoint they may be described as follows: Areas of "aseptic necrosis," often with sequestration, occur in certain sites of predilection. These are the head of

FIG. 10. Localized areas of apparently increased density in the head of the humerus of a patient carrying a body burden of 1.6 μg.

FIG. 11. Areas of increased density in the proximal tibia and fibula in patient carrying a body burden of 4.2 μg. of radium.

the femur (Fig. 14), the mandible (Fig. 15), tarsal scaphoid (Fig. 16), and vertebral bodies (Fig. 17). Occasionally one observes necrosis with sequestration in portions of the head of the radius (Fig. 18) and the superior aspect of the acetabulum (Fig. 19). Fragmentation of the patella (Fig. 20) is not infrequently seen. The dynamics of the skeleton draw attention to a correlation between these sites of vulnerability for bone necrosis and the areas that, during normal activity, regularly undergo greatest mechanical stress. In the pathogenesis of this type of bone lesion, the association of radium deposited in bone and repeated mechanical insult seems apparent. Taken by itself, the appearance of one of these areas of "aseptic necrosis" in an otherwise apparently "healthy" adult is *not diagnostic* of radium poisoning, *but* is suggestive.

Fig. 12. Areas of increased density in the tarsals and metatarsals of a patient carrying a body burden of approximately 10 μg. of radium.

Fig. 13. Note the loss of the normal trabecular pattern in the proximal end of the tibia in a patient carrying a total body burden of 10.7 μg.

CLINICAL HISTORIES

The clinical histories of those patients who received radium therapeutically and those of the 6 "dial painters" are illustrative of the progress of the disease over a long period; the appearance of the lesions after a long latent period, and of the lack of realization by many of these individuals that they had been previously given or carried a body burden of radium.

Patient 119. White male, aged thirty. Total body burden 0.03 μg. This man is the brother of Patient 120 and received radium orally from the age of six to eight years. He has

FIG. 14. "Aseptic necrosis" with sequestration of the superior aspect of the head of the right femur in a patient carrying a total body burden of 0.7 μg. of radium. Roentgenography done three years after this examination demonstrated identical destruction of the head of the other femur.

been asymptomatic. Complete physical examination and roentgenographic study of the skeleton revealed no body lesions. The teeth do not demonstrate lesions similar to those present in his brother.

PATIENT 311. White female, age unknown. Total body burden 0.06 μg.

The patient worked as a radium dial worker in Ottawa, Illinois, from 1941 to 1944, and for six months in 1946. The clinical history, physical examination, and skeletal roentgenographic survey were all negative.

PATIENT 115. White female, aged seventy-two. Total body burden 0.06 μg.

In 1939, the patient received intramuscular injections of radium chloride in the lower back for "arthritis of the lower spine." There was a negative history and no other parts were involved. The radium was given intramuscularly every other day, later reduced to once weekly until a total of 10 to 15 injections had been given. The quantity of radium administered per injection is not known. Within one month, the patient noted marked relief from pain, and, within six months, the pain had subsided. She has remained asymptomatic. The history and physical examination gave essentially negative data.

PATIENT 109. White female, aged sixty-two. Total body burden 0.06 μg.

In approximately 1930, the patient was prescribed radium water orally for a number of weeks for "sacroiliac pain." The patient states that spontaneous "black and blue marks" have occurred intermittently for years, and that she has noted prolonged bleeding following skin lacerations. The teeth had to be removed with care because of this bleeding tendency. Her mother and one other sister had similar difficulty with prolonged bleeding. The dental examination revealed an absence of all teeth. Skeletal roentgenographic examination was negative for bony lesions related to radium poisoning.

PATIENT 303. White female, aged fifty. Total body burden 0.11 μg.

The patient was employed as a radium dial painter in Ottawa, Illinois, for six months in

1922. In 1934 she noted the onset of an intermittent sharp pain in the hands that would last for one to two days and then subside for two to three months. Gradually, this type of pain also involved the shoulders, and by 1950, the hips. The patient also complained of difficulty in walking. During 1951, a sharp, aching pain developed in the long bones, which was increased on pressure over the bones.

The general physical examination and the complete skeletal roentgenographic survey were completely negative. The relationship of the pain in the bone to the presence of radium is difficult to establish in view of the negative roentgenographic findings.

Patient 120. White male, aged thirty-two. Total body burden 0.15 μg.

The patient had been in excellent health all of his life until the age of thirty-two years at which time his oral surgeon noted a resorption of the teeth at the gum line leading to the fragmentation and loss of the crown of a central incisor. Roentgenography of the teeth demonstrated a bizarre appearing widening of the pulp cavity and destruction of the dentin arising within the teeth.

Fig. 15. Necrosis of the mandible in patient carrying body burden of 1.6 μg. of radium.

Fig. 16. Localized area of bone necrosis in the tarsal scaphoid of a patient carrying a body burden of 1.3 μg. Also note small typical areas of bone resorption in the distal tibia and fibula.

Dr. W. W. Wainwright of the School of Dentistry of the University of Illinois saw the patient in consultation and recognized these changes as similar to those that had been observed in radium dial paint poisoning cases as well as in some of the patients in the Elgin Hospital series. The patient was referred to our group for study.

On questioning, the patient at first did not recall ever having received radium, and because of his age it was difficult to conceive that he had been given radium therapeutically at the height of its popularity as a medicament. A perusal of the old files of Schlundt revealed, however, that the patient's father, a physician, had been a patient of Dr. . . . (Patient 110) and had been

FIG. 17. Destruction of vertebral bodies in patient carrying body burden of 10.7 μg. of radium.

given radium by him. The patient then remembered that for a period of about two years, when he was between the ages of eight and ten, his father had had his brother, mother, and himself each drink a glassful of "radium water" daily as a "tonic."

The general physical examination, hematologic studies, and roentgenographic examination of the entire skeleton were completely negative except for the destructive lesions in the teeth that are suggestive of radium poisoning.

PATIENT 305. White female, aged forty-nine. Total body burden approximately 0.2 μg.

The patient worked as a radium dial painter in Ottawa, Illinois, during 1924. In 1935 she noted the onset of pain in the hands, gradually also involving the wrists, elbows, shoulders, and left knee. The pain was occasionally accompanied by redness, swelling, and stiffness of the joints. There has been little progression of the symptoms over the years.

A complete roentgenographic skeletal survey demonstrated joint changes, consistent with a diagnosis of rheumatoid arthritis. The changes in the hands suggested gouty arthritis. It is considered likely that the clinical condition is not related to the deposition of radium in this patient.

PATIENT 121. White male, aged approximately seventy-two. Total body burden 0.25 μg.

This individual, who has been asymptomatic, has been a radium chemist for the last forty years. While visiting the Argonne National Laboratory, he kindly consented to a determination of his total body burden and a complete skeletal roentgen study. The skeletal survey revealed no bony abnormalities. The total body burden of Patient 121 had been de-

FIG. 18. Necrosis and sequestration of the head of the radius in a patient carrying a body burden of 1.0 μg. of radium.

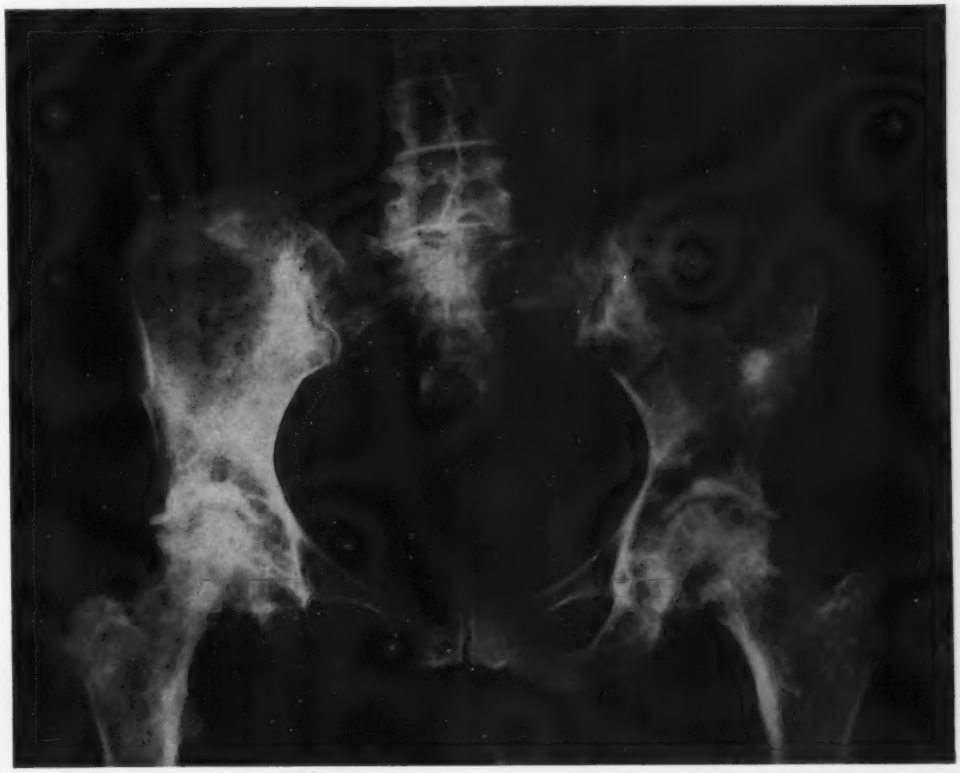

Fig. 19. Multiple lesions in the pelvis of a patient carrying a body burden of 4.2 μg. Note the destruction of the heads of both femora and the acetabula, the loss of joint space, change in trabecular pattern, and the localized areas of increased density in the pelvis and femora.

termined by the Aub group[4] and is listed in their series as Patient 28.

Patient C. White female, aged forty-seven. Total body burden 0.4 μg.

The patient was admitted to the Elgin State Hospital on February 28, 1931, with a diagnosis of schizophrenia. During 1931, she received 10 μg. of radium weekly by the intravenous route to a total dosage of 250 μg.

At the time that the radium was given, the teeth were reported to be in poor condition. The condition of the teeth deteriorated markedly during the following years, and the patient was completely edentulous when she was examined in 1951. The general physical examination was essentially noncontributory.

Roentgenography of the entire skeleton revealed minimal but definite areas of bone resorption in the humerus, radius, ulna, tarsals, metatarsals, phalanges, tibia, and fibula, and moderately severe changes in the shafts of the femurs.

This patient demonstrates diagnostic evidence of bone damage at a body burden of 0.4 μg. She is presumed to have received radium uncontaminated with mesothorium or radiothorium.

Patient 117. White male, aged forty-three (physician). Total body burden 0.5 μg.

At the age of seventeen years, the patient had poliomyelitis and, as therapy for this, received an unknown amount of radium parenterally in several injections from his physician (Patient 110). He was left with residual atrophy of the lower extremities but has had no symptoms referable to radium poisoning.

Roentgenography of the skeleton revealed minimal to moderately marked areas of bone resorption in the cortices of the femur, tibia, fibula (Fig. 21), metatarsals, metacarpals, and the phalanges.

Patient B. White male, aged fifty-one. Total body burden 0.5 μg.

In 1931, the patient, who was then twenty-seven years old and hospitalized at the Elgin

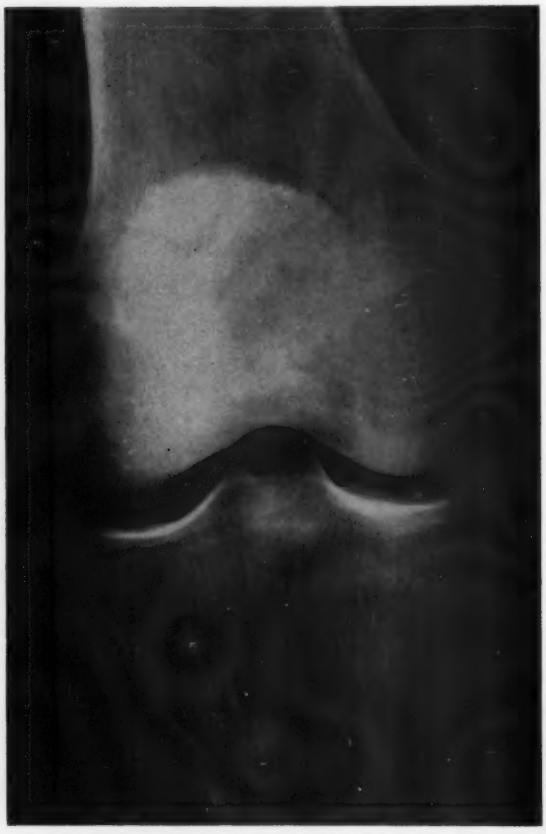

FIG. 20. Fragmentation of the patella in a patient carrying a body burden of approximately 10 μg. of radium.

State Hospital for schizophrenia, was administered 10 μg. of radium intravenously each week to a total dosage of 150 μg.

When seen in May, 1951, the general examination was within normal limits. Roentgenography of the entire skeleton revealed only a few small areas of bone resorption in the fibulae.

PATIENT 101. White female, aged seventy-one. Total body burden 0.5 μg.

In June, 1918, at the age of thirty-seven years, the patient had her first episode of "arthritis." This involved the wrists, shoulders, neck, and knees and lasted about a year. She then had recurrent episodes of "arthritis," and in 1926 and 1927, a physician (Patient 110) gave her radium intramuscularly in an effort to alleviate the condition; apparently with some temporary relief of pain. Episodes of pain have continued through the years, and there has occurred gradual limitation of motion of the joints of the hands, wrists, knees, and hips.

Physical examination revealed a blood pressure of 180/90. There was some weakness of the upper extremities. The joints of the hands were moderately deformed with some limitation of motion but with an almost complete range of motion of the hip and knee joints; the ankle joint had about 20–30° limitation.

Roentgenography showed rather marked arthritic changes in most of the joints. Minimal areas of bone resorption were noted in the fibulae.

PATIENT PAUL N. White male, aged forty-six. Total body burden 0.5 μg.

The patient was admitted to the Elgin State Hospital in 1933 and was injected intravenously with 10 μg. of radium at weekly intervals to a total dosage of 110 μg. The history during the ensuing years was essentially negative. A dental examination in 1938 revealed that the teeth were in good condition.

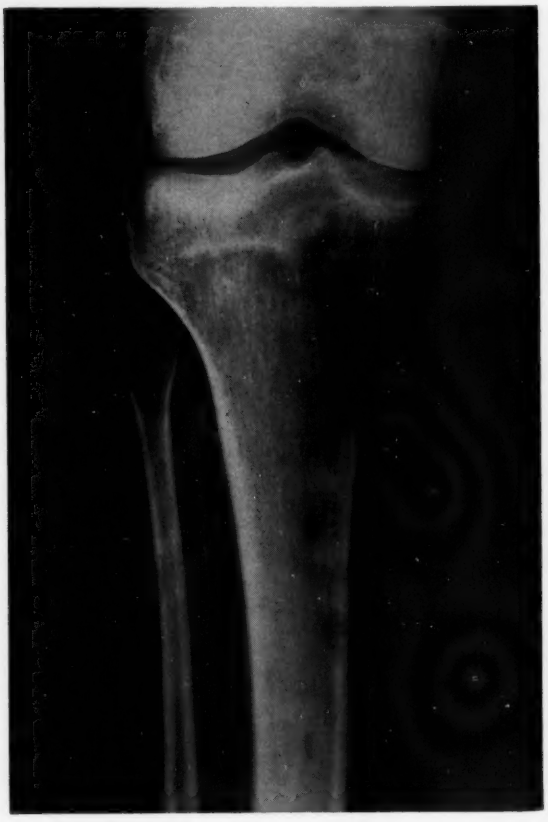

FIG. 21. Patient 117. Body burden 0.5 μg. Areas of bone resorption in the tibia and fibula in an asymptomatic person who received radium injections at the age of seventeen years.

The patient was examined in August, 1950, and the general physical examination revealed findings within the limits of normal except for severe pyorrhea and the presence of only a few carious tooth stumps.

Roentgenography of the skeleton showed a few typical minimal areas of bone resorption in the tibiae, fibulae, and humeri. Roentgenography of the entire skeleton was again done during the summer of 1953 and revealed no progression in the extent of the lesions.

Patient 309. White female, aged 46. Total body burden 0.52 μg.

When she was sixteen years old, the patient worked as a dial painter for one year. At this time, it was a common practice to point the radium-containing brush with the lips; and this the patient did.

In 1932, approximately eleven years after the time of ingestion of radium, she developed pain in the region of the left elbow that progressed. After three to four months, a golf ball size mass was found in the antecubital space. The arm was disarticulated at the shoulder at the Cook County Hospital, Chicago, Illinois, and a diagnosis of sarcoma was made on the basis of a microscopic examination of the tumor tissue. The original specimen was located in the Pathology Museum of the Cook County Hospital in 1952 and, through the courtesy of Dr. Hans Popper, was made available to this group for study. The original histopathologic diagnosis of fibrosarcoma was confirmed. Roentgenography of the amputated arm revealed the characteristic typical areas of resorption in the radius, ulna, and humerus, as well as the osteolytic tumor of the head of the radius (Fig. 22).

Prior to the amputation, the patient had borne 3 normal children. Since the amputation of the arm, the patient has been in good health except for intermittent pain in the left ankle and the right knee.

Physical examination was noncontributory. Roentgenography revealed small areas of bone resorption in the skull, humerus, and femur. Moderately marked bone resorption and necrosis were present in the metatarsals, tibia, and fibula. One tarsal scaphoid demonstrated a typical moderately advanced bone necrosis.

Patient U. White female, aged forty-six. Total body burden 0.6 μg.

The patient was first admitted to the Elgin State Hospital in September, 1931, with a diagnosis of dementia praecox. The following November she received a course of malarial therapy with no apparent benefit. No notes were present in the patient's hospital chart relating to the administration of radium. However, in the protocol sheets of Schlundt *et al.* were found references to the administration, in 1931, of 10 μg. of radium per week intravenously to a total dose of 90 μg.

In 1932, the patient complained of vague

Fig. 22. Patient 309. Body burden 0.52 μg. Fibrosarcoma of the head of the radius. Roentgenogram made on anatomical specimen of arm amputated in 1931.

joint pains, but since then has not had a recurrence of these symptoms.

The general physical examination revealed nothing unusual beyond the presence of a few carious teeth.

PATIENT S. White female, aged seventy-four. Total body burden 0.6 μg.

In 1931, at the age of fifty-one years, the patient, who was a schizophrenic hospitalized at the Elgin State Hospital, was given intravenously 10 μg. of radium per week to a total of 70 μg.

A physical examination done in August, 1950, revealed a senile, withdrawn, white female who was completely edentulous. Only those changes that one would expect in a patient of about this age were seen. There were no obvious orthopedic deformities, and the joints moved freely.

Roentgenography of the entire skeleton revealed typical minimal lesions in the thoracic and lumbar vertebrae and in the sacrum. There were a few small areas of bone resorption in the fibulae. The entire skeleton was roentgenographed again during the summer of 1953. There was no progression in the extent or numbers of lesions during this three year period.

PATIENT 306. White female, aged forty-seven. Total body burden 0.72 μg.

This patient worked as a dial painter in Ottawa, Illinois, from 1923 to 1934. For the first two to three years she tipped the brush with her lips, but this practice was discontinued, and a glass pencil was used thereafter. The patient has been in good health during the years since her employment.

The general physical examination was negative. Roentgenographic survey revealed the typical minimal lesions in the fibulae that are diagnostic of the presence of radium in the skeleton.

PATIENT 116. White female, aged fifty-three. Total body burden 0.7 μg.

At the age of twenty-eight years, the patient received 12 injections of radium chloride as therapy for a mental depression that followed the birth of her first child. Six years later, she was given 4 more injections for a recurrence of the depression after the birth of a second child. The physician who administered the medication informed us that each dose contained 10 μg. of radium chloride.

In December, 1948, the patient first noted pain in the right groin, especially after much walking. During the ensuing few months, the pain spread throughout the entire right lower extremity and was associated with stiffness of the right hip and knee joints. In June, 1950, the patient consulted Dr. H. A. Sofield, who referred her to Dr. R. M. Potter for roentgenography. His study revealed an area of aseptic necrosis in the head of the right femur that was suggestive of the bone changes appearing secondary to the presence of radium deposited in the skeleton. Roentgenography in 1954 demonstrated the development of an identical area of necrosis of the head of the other femur.

PATIENT I. White female, aged fifty-one. Total body burden 0.8 μg.

The patient was admitted to the Elgin State Hospital in December, 1930, with a diagnosis of schizophrenia. During 1931, the patient was given intravenously 10 μg. of radium per week to a total of 180 μg.

In the ensuing years, the patient had no orthopedic complaints. Her teeth remained in fairly good condition, and a dental examination in January, 1951, revealed that the patient had only a few carious areas. A general physical examination was made in May, 1951. There were no general findings of note. The orthopedic examination was normal. At the same time, roentgenography of the entire skeleton demonstrated no bony abnormalities. In the summer of 1953, the roentgenographic skeletal survey was repeated and again revealed no changes.

PATIENT Q. White male, aged fifty-six. Total body burden 0.8 μg.

The patient was admitted to the Elgin State Hospital in April, 1918, with a diagnosis of schizophrenia. During the early months of 1931 he was given intravenously 10 μg. of radium per week to a total dose of 450 μg. In the ensuing time, he developed no symptoms referable to the presence of radium in his skeleton. In 1948, the patient was engaged in two altercations in the ward and suffered a fracture of the nasal bone and a fracture of the medial malleolus of one ankle.

The general physical examination revealed an emaciated, markedly deteriorated male of about the stated age. Only a few stumps of teeth remained and severe pyorrhea was present. The rest of the findings were within normal limits. There were no orthopedic deformities. A complete skeletal roentgenographic survey

was done, and the only finding was the presence of a few small areas of bone resorption in the fibulae. The complete skeletal survey was repeated in the summer of 1953 and demonstrated no progression either in the size or number of lesions present.

Patient Edith C. White female, aged forty-six. Total body burden 0.8 μg.

The patient was admitted to the Elgin State Hospital in June, 1931, with a diagnosis of schizophrenia. During the month of admission, radium treatment was begun. The material was administered intravenously at weekly intervals in dosages of 10 μg. to a total of 260 μg. The patient was discharged from the hospital in December, 1931, but was re-admitted in October, 1948, with a diagnosis of involutional psychosis. She remained until January, 1949. The patient was married in 1932, and children were born to her in 1933, 1934, and 1936. All the children are living and are healthy. During the interval from 1931 to 1951 the patient gave no history of pain in the joints, fractures, or anemia.

A general physical examination was not obtained. A total skeletal roentgenographic survey was made in 1951. This gave no evidence for the presence of radium in the skeleton.

Patient 105. White female, aged seventy-four. Total body burden 0.86 μg.

This patient did not know she had received radium. This fact was discovered when the roentgenograms of her husband, known to have been given radium, were reviewed by Dr. J. W. Clark of the Presbyterian Hospital, Chicago, Illinois, who had seen her films on a previous occasion. The similarity of the skeletal changes in the two patients led to the conclusion that the etiological factors might be identical. The patient was referred to this group for study, and the presence of a total body burden of 0.8 μg. was demonstrated.

In 1947, the patient struck the lower part of her right leg and, because of the continued pain, swelling and lameness, sought medical aid. A roentgenogram of the tibia was made (Fig. 23). The bone changes were interpreted to be the result of osteomyelitis, and a course of 10 treatments of external roentgen irradiation was given. The improvement of the condition following roentgen therapy suggests that the diagnosis in actuality could be Ewing's tumor or multiple myeloma.

Fig. 23. Patient 105. Body burden 0.86 μg. "Osteoid osteoma" of the tibia which later metastasized extensively to the patient's lungs. Also note other typical changes in the patella, tibia and fibula.

In 1948, a biopsy of the tibia was done, and a diagnosis of osteoid osteoma was made. At a later date, the lesion was curetted and was considered to be a giant cell tumor. There was little discomfort following the curettement; however, the patient was unable subsequently to bear weight on the leg. In 1949, a pathological fracture was sustained, and a high amputation of the leg was performed because of the extensive bone involvement and absence of bony union.

The patient was examined by the authors on February 19, 1952. She was a small, thin, alert female who showed marked evidence of chronic illness and weight loss. No pain or discomfort in any of the joints had been experienced since 1949. She had noted pain in the mid-portion of her back to the right of the midline since January, 1952.

Roentgenographic examination revealed multiple areas of increased density throughout both lung fields. These were suggestive of metastatic sarcomatosis. Osteolytic lesions were present in the ninth rib on the right side.

Shortly thereafter the patient suffered a cerebral accident either as a result of vascular disease or metastasis of tumor to the brain. The patient failed gradually and died on October 8, 1952, from generalized sarcomatosis. Unfortunately, an autopsy was not performed.

PATIENT K. White female, aged forty-five. Total body burden 0.9 μg.

The patient was admitted to the Elgin State Hospital in January, 1929, with a diagnosis of schizophrenia. In 1931, she was given radium intravenously in 10 μg. doses at weekly intervals to a total of 200 μg. On March 25, 1933, the patient was reported to have a marked swelling of the right mandible, which seemed to be caused by an infected tooth. All dental reports subsequently have been essentially negative. The patient was examined in May, 1951. Her teeth were reported to be in very poor condition; they were almost all worn off to the gingival margin and were extremely carious. The remainder of the general physical examination was essentially negative. There was no limitation of motion of any of the joints nor was pain elicited on motion of the joints. The complete roentgenographic skeletal survey was done in May, 1951, and revealed moderately marked resorption in the radii and minimal resorption in the ulnae. The other bones demonstrated no typical changes. A complete roentgenographic survey of the total skeleton was repeated during the summer of 1953. There was no progression of the lesions previously found nor were other bones affected.

PATIENT T. White female, aged forty-one. Total body burden 1.0 μg.

This patient was admitted to the Elgin State Hospital in July, 1931, with a diagnosis of schizophrenia. In 1931, she was given 10 μg. of radium intravenously per week to a total of 110 μg. The patient had had no orthopedic complaints during the years of hospitalization. On examination in August, 1950, it was noted that the teeth were in very poor repair; all were carious and many were missing. Very severe pyorrhea was present. The remainder of the general physical examination was not remarkable. There was no limitation of motion in any joints. No bone lesions typical of radium poisoning were revealed in the skeletal roentgenograms.

PATIENT J. White female, aged fifty-four. Total body burden 1.0 μg.

The patient was admitted to the Elgin State Hospital in August, 1930, with a diagnosis of schizophrenia. Beginning in January, 1931, the patient was given 10 μg. of radium intravenously at weekly intervals to a total dose of 200 μg. A general physical examination in August, 1950, revealed a well developed, well nourished white female in good contact with her environment. The teeth were in fair repair; several were missing but those remaining had only a few cavities. There was moderate scoliosis of the thoracic spine to the right at the level of D8–12. There was no pain on percussion over this area, nor was there limitation of motion. The remainder of the general physical examination was essentially negative. There was no other evidence of orthopedic disease except for the presence of an old healed fracture of the left elbow, which had been sustained during childhood. Complete skeletal roentgenography demonstrated numerous small areas of bone resorption and coarsening in the trabecular pattern in the radii and ulnae and in the tibiae and fibulae. Minimal lesions of bone resorption were present in the femur. A complete skeletal roentgenographic survey in the summer of 1953 revealed no progression in the bony lesions since the 1951 examination.

PATIENT 113. White male, aged fifty-seven. Total body burden 1 μg.

During 1925 through 1936, the patient received an unknown number of injections of radium from his physician (Patient 110) for the treatment of psoriasis. Five years ago, he developed pain in the right elbow, especially intense while playing golf. Roentgenography of the elbow in January, 1950, revealed "aseptic necrosis" of the head of the right radius.

In April, 1951, the total body burden of radium was determined to be 1.0 μg. Roentgenography revealed changes in the trabecular pattern of the thoracic and lumbar vertebrae, minimal to moderately marked areas of bone resorption ("streaking") in the ulna, femur, tibia, and fibula in addition to the previously described changes in the head of the right radius. The psoriasis is still present.

Patient O. White female, aged forty-eight. Total body burden 1.0 μg.

The patient was admitted to the Elgin State Hospital in March, 1931, with a diagnosis of schizophrenia. During 1931, she received 10 μg. doses of radium intravenously at weekly intervals to a total of 260 μg. According to the records of the Elgin State Hospital, the patient complained, in 1940, of pain in the left hip. At this time, roentgenograms of the pelvis, the lower sacral spine, and ankle revealed no change. No further record of the course of the condition was found. A total skeletal roentgenographic study in May, 1951, demonstrated moderately marked areas of resorption in the radii and minimal areas of resorption in the humeri and fibulae. The skeletal study was repeated in the summer of 1953 and the findings were identical. There was no progression in the lesions.

Patient 313. White female, aged forty-eight. Total body burden 1.3 μg.

The patient had been employed as a dial painter in Ottawa, Illinois, since 1924, with the exception of one year, until her illness in 1953. During the first two years of employment, the patient tipped the brush with her lips.

The history was uneventful until 1948, at which time she tripped on a curbing and fractured the shaft of the left femur. Four months later, the femur was refractured in another fall. One year after the initial fall, the patient returned to her employment. In October, 1951, the patient was seen routinely with other former employees of the Ottawa, Illinois, radium dial plant. Roentgenographic changes typical of radium necrosis and resorption were present in the skull, head of the left femur, radii, ulnae (Fig. 24), left tibia, left fibula, and left metatarsals. No tumors were present, nor was there evidence of change in the pelvis (Fig. 25).

During the ensuing years, no changes in the state of her health occurred until January, 1953,

Fig. 24. Patient 313. Body burden 1.3 μg. Extensive areas of bone resorption in the radius and ulna.

when a persistent, severe pain deep in the right buttock developed and continued unabated. Pelvic examination by her physician, in 1953, revealed a carcinoma of the cervix. This was treated by radium needle implantation and pelvic roentgen irradiation. A few months later, a total hysterectomy was performed because of the presence of fibroid tumors in the uterus.

Roentgenography of the pelvis in September,

FIG. 25. Patient 313. Body burden 1.3 μg. Note absence of typical changes from radium deposition in roentgenogram of pelvis made in 1951.

1953, revealed an expansile osteolytic lesion in the right ischial tuberosity that also involved the pubis (Fig. 26). This was presumed to be a metastasis from the cervical carcinoma, and the patient was given approximately 2,000 rep of roentgen radiation directed to the lesion.

The patient was then referred, in October, 1953, to this group for further study. It seemed apparent that the lesion did not resemble a metastasis, but rather an osteogenic lesion. Dr. C. H. Hatcher of the University of Chicago Clinics performed a biopsy, which revealed a fibrosarcoma (Fig. 27). Because a local resection was impossible, a right hemipelvectomy was done.

The patient re-entered the Argonne Cancer Hospital in May, 1954, with a local recurrence of tumor at the amputation site and died on July 22, 1954.

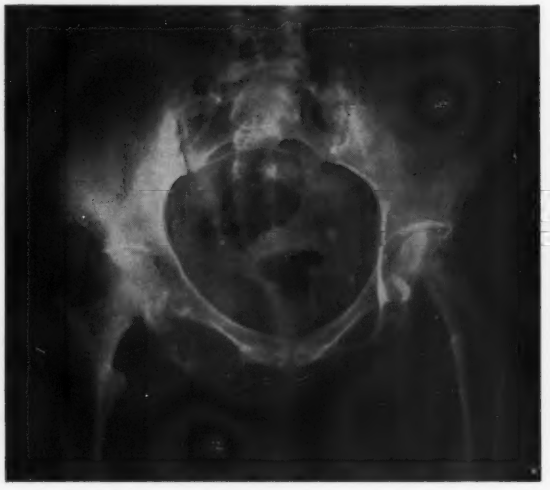

FIG. 26. Patient 313. Body burden 1.3 μg. Note the fibrosarcoma of the right ischium and pubis present on roentgenograms made two years after previous roentgenogram which shows no typical changes.

PATIENT 112. White female, aged forty-eight. Total body burden 1.3 μg.

At the age of twenty-one, the patient was hospitalized with a vague illness diagnosed as "subacute bacterial endocarditis" and "polyarthritis" and at this time was confined to bed for two years. During the next few years, intermittent stiffness of the joints of the hands, shoulder, and lower back was present. For this, the patient's physician prescribed a solution of a soluble salt of radium, of unknown concentration, to be taken orally. The dosage is unknown. All that is known is that the patient took 15 to 30 drops of the solution once or twice daily for several weeks.

In July, 1947, about nineteen years after the

FIG. 27. Patient 313. Body burden 1.3 μg. Surgical specimen following hemipelvectomy demonstrating fibrosarcoma of the ischium invading the acetabulum.

administration of radium, the patient was suddenly affected with a crushing type of pain of great severity in the left foot. She was admitted to Billings Hospital of the University of Chicago Clinics on February 3, 1948, where roentgenography of the entire skeleton was done and interpreted by Dr. P. C. Hodges. The examination revealed a large, irregular cavity in the body of the tarsal scaphoid of the left foot of undetermined nature (Fig. 28). There was an unidentified negative shadow in the left occipitoparietal region of the skull. Minimal grade aseptic necrosis of the head of the left humerus and both glenoids was noted. An anthropoid type of spongiosa in the lumbar vertebrae, pelvis, and upper ends of the femur, was observed. Its significance was not known at that time.

The patient was seen in consultation by one of the authors (A.M.B.), and determinations of breath samples in Dr. Norris' laboratories at the Argonne National Laboratory revealed significant amounts of radon, diagnostic of radium deposits in the body. At his suggestion, she was placed on a low calcium, low phosphorus diet, and was given ammonium chloride and parathormone, until she was discharged on March 8, 1948. Although a measurable increase in radium excretion was observed as a result of this regimen, there was no roentgenographic evidence of bone healing. The peripheral blood picture at this time was not remarkable, but a study of the sternal marrow gave distinct evidence of overactivity. There was moderate erythroblastic activity without shift to the left, but it was the opinion of Dr. M. H. Block that more mitoses than usual were present. The blood picture was not that of a "radium aplasia."

Fig. 28. Patient 112. Total body burden 1.3 μg. Roentgenogram reveals area of necrosis of tarsal scaphoid in 1948.

Fig. 29. Patient 112. Widespread sarcomatosis of the same foot when re-roentgenographed in 1951. There is complete destruction of the scaphoid and involvement of the other tarsals.

Pain on walking increased after the patient was discharged from the hospital. The onset of pain in other bones occurred in 1950 and later became generalized throughout the skeleton.

In January, 1951, determination of radium content of the body was estimated to be 1.3 μg. by combined gamma-ray and radon methods. Roentgenography of the skeleton demonstrated a progression of all lesions, especially in the left foot where destruction of the scaphoid was complete and partial destruction involved other tarsals (Fig. 29). A biopsy of the scaphoid was done by Dr. F. Chandler, and a diagnosis of fibrosarcoma was made (Fig. 30). On February 20, 1951, the left leg was amputated 15 cm. below the knee. The patient died of generalized sarcomatosis on August 15, 1951, twenty-three years after the administration of radium and four years after the onset of the symptoms. During the last three years, the number of "punched-out" lesions in the skull approximately doubled (Fig. 31).

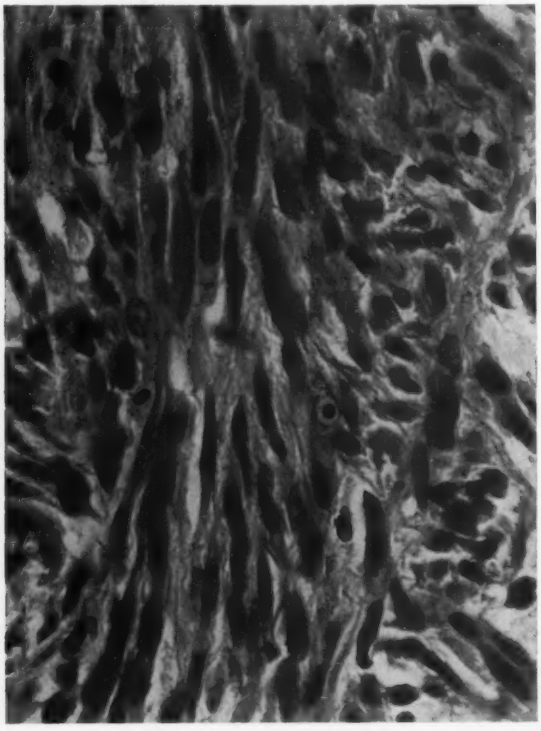

FIG. 30. Patient 112. Anaplastic fibrosarcoma arising in the tarsal scaphoid.

PATIENT A. White female, aged forty-six. Total body burden 1.6 μg.

This patient was admitted to the Elgin State Hospital in May, 1930, with a diagnosis of schizophrenia. In 1931, she was given 10 μg. of radium per week intravenously to a total of 150 μg. During 1940, it was noted that the patient seemed to have a peculiar gait, and roentgenograms were made of both femora. These revealed an "old osteochondritis—no fractures." The patient was seen by the authors during August, 1950, and was observed to have a waddling gait, with little apparent discomfort.

General physical examination revealed nothing significant except for moderate limitation of motion of the hips. A complete skeletal roentgenographic survey demonstrated marked destruction of the heads of the femora. There was minimal mottling of the heads of the humeri, and there were minimal areas of bone resorption in the tibiae and fibulae. These destructive processes in the heads of the femora were identical with those observed in other radium patients and did not resemble "old osteochondritis."

The complete skeletal roentgenographic survey was repeated during the summer of 1953 with the following findings: There was increased destruction of the heads of both femora, most marked on the left. The acetabula were also increasingly involved in this destructive process. There was no increase in the size or number of bony lesions in the rest of the skeleton.

PATIENT 114. White female, aged fifty-six. Total body burden 1.6 μg.

The patient, the wife of a physician, did not remember receiving radium. In the winter of 1950, her dentist noted a pinkish discoloration of the teeth and advised the patient that this phenomenon had been described and associated with radium poisoning. She was referred by her husband for a determination that revealed a total body burden of 1.6 μg.

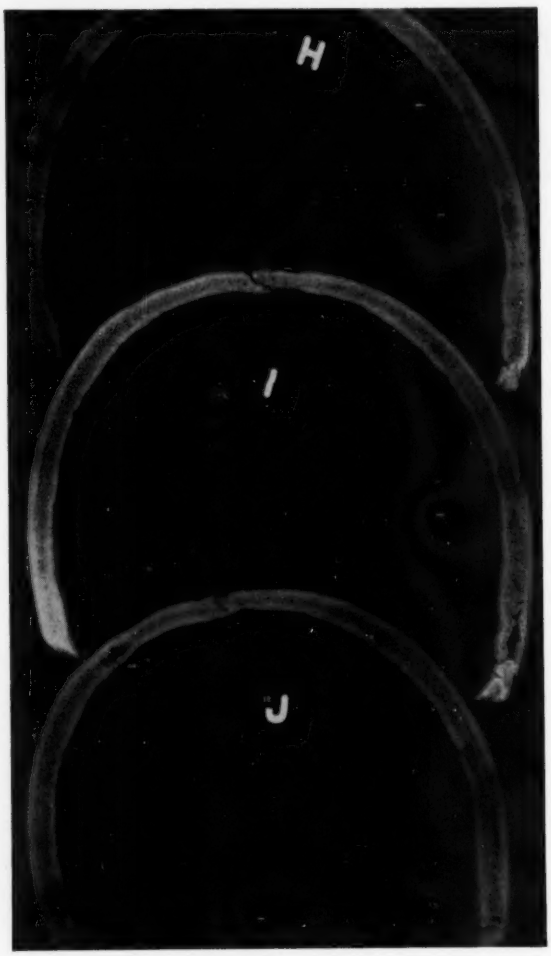

FIG. 31. Patient 112. Postmortem coronal sections of the skull demonstrating areas of bone destruction visible on the roentgenograms as "punched out" areas.

Roentgenography demonstrated minimal trabecular changes in the thoracic and lumbar vertebrae; minimal changes of bone resorption and necrosis in the ilium and acetabulum; and similar moderately marked changes in the humerus, radius, ulna (Fig. 32), tibia, and fibula. The patient is asymptomatic.

Patient 102. White male, aged eighty-one. Total body burden 1.6 μg.

In March, 1939, the patient noted a small spicule of bone protruding from the gum at the base of a left lower molar. This was accompanied by slight tenderness, redness, and swelling in the same region. He entered Billings Hospital on the service of Dr. D. B. Phemister. From that time until October, 1940, the patient had intermittent drainage from and sequestration of the left ramus of the mandible. On June 17, 1941, the defect of the mandible was repaired with a bone transplant from the right ilium. On October 5, 1941, the infected necrotic bone graft was removed.

The patient does not remember ever having received injections of radium or having taken radium water by mouth. However, since he was the husband of Patient K.B., who was a known "radium poisoning" case, Dr. Phemister referred the patient to the authors for study.

Determination revealed the presence of 1.6 μg. of radium in the body. A review of films made in 1941 and 1942 in the Department of Radiology of the University of Chicago Clinics demonstrated the mandibular defect, moderately severe areas of bone resorption ("streaking") in the fibula and radius, and minimal, similar lesions in the tibia and ulna.

Fig. 32. Patient 114. Body burden 1.6 μg. Multiple typical areas of bone resorption in the shafts of the radius and ulna in an asymptomatic person who was unaware that she had received radium.

Patient N. White female, aged fifty-four. Total body burden 1.8 μg.

This patient was admitted to the Elgin State Hospital in May, 1931, with a diagnosis of schizophrenia. During September, 1931, the intravenous administration of 10 μg. of radium was begun and continued each week to a total dose of 360 μg. The patient was examined during August, 1950. At that time there were only 5 lower central teeth in the mouth. These were in fair repair. Nothing unusual was revealed during the general physical examination. No orthopedic defects were observed. Complete skeletal roentgenography was done during the summer of 1950, and the typical minimal lesions were seen in the humeri, fibulae, and radii. A complete skeletal survey was repeated during the summer of 1953 and revealed no progression in the bone lesions.

Patient Rose D. White female, aged forty-five. Total body burden 1.9 μg.

The patient was admitted to the Elgin State Hospital in December, 1931, with a diagnosis of manic-depressive psychosis, manic type. Over the years, the patient has had approximately 15 admissions to the hospital to the present time. The manic phase usually lasts about three months, then she is able to resume a sheltered life at home with her parents between episodes. The physical examination on the first admission to the Elgin State Hospital and on subsequent admissions has been essentially negative. During 1931, the patient was given intravenously 10 μg. of radium per week to a total of 200 μg.

An admission note made during 1940 records

FIG. 33. Patient 111. Body burden 4.2 μg. Loss of normal trabecular markings and areas of increased density in the heads of the femora, pelvis, sacrum and lumbar vertebrae.

that the patient complained of pain in the left leg. A progress note dated December 20, 1941, mentions that the patient was attacked by another patient and sustained a fracture of the nasal bones and a possible fracture of the femur. Which femur was involved was not specified, and no roentgenograms are available for examination. On September 24, 1949, a note was made in the patient's chart as follows: "Enjoys telling about her somatic difficulties particularly her arthritis and how doctors told her not to wear high heels." A roentgen examination of the pelvis and right hip was done on September 30, 1949, at the Elgin State Hospital and was reported as follows: "The heads of the right femur and the acetabulum are markedly deformed, with an obliteration of the articular separation, so that the motion of the joint is markedly limited. The surrounding osseous structures are of increased opacity."

The patient was examined during 1951, and the general physical examination revealed nothing unusual. Complete skeletal roentgenography done in June, 1951, demonstrated the following changes. The tibiae, fibulae, radii, and ulnae all had the typical areas of bone resorption to a marked degree. The shaft of the femur showed pronounced changes in trabecular pattern as well as multiple areas of bone resorption. At least three thoracic vertebrae showed destruction of the bodies with collapse of these vertebral bodies. Both tarsal scaphoid bones showed small areas of bone resorption. There was a destruction of the superior surface of the head of the right femur with flattening and a destruction of the adjacent acetabulum. The joint space was obliterated. The adjacent bone showed a disturbance in the trabecular pattern.

PATIENT P. White female, aged forty-seven. Total body burden 2.6 μg.

This patient was admitted to the Elgin State Hospital during February, 1931, with a diagnosis of schizophrenia. During that year she received radium intravenously in 10 μg. doses at weekly intervals to a total of 250 μg.

At the time of radium administration, the teeth were found to be in extremely poor condition. They deteriorated markedly during the following years, and at the time of examination in 1950, the patient was edentulous.

The general physical examination that was done during October, 1950, revealed no clinical findings of note. A complete skeletal roentgenographic study that was done during 1951 revealed necrosis and destruction of the head of the left humerus. The glenoid processes of the left scapula also demonstrated necrosis and destruction. There was marked diminution in the joint space. The shafts of the femora had the typical linear areas of bone resorption. There was a questionable narrowing of the joint space between the vertebral bodies of the sixth and seventh thoracic vertebrae.

PATIENT R. White female, aged fifty-six. Total body burden 3.6 μg.

The patient was admitted to the Elgin State Hospital in January, 1930, with a diagnosis of schizophrenia. In 1931, she received intravenously 10 μg. of radium each week to a total of 410 μg.

In 1933, the clinical notes record the condition of her teeth as being good. In 1937, pyorrhea was discovered on annual dental examination. By 1950, she was completely edentulous. No significant clinical findings were found when a complete physical examination was done during August, 1950.

Complete skeletal roentgenography was carried out during May, 1951. There were numerous areas of bone resorption in the tibae, fibulae, radii, and ulnae. The humeri demonstrated a few areas of bone resorption and several areas of apparent increased density in their heads. Both tarsal scaphoids demonstrated small areas of bone resorption. The skeletal study was repeated during the summer of 1953. There was no progression in the num-

ber or severity of the bone lesions as judged by a comparison with films made two years earlier.

Patient 111. White female, aged sixty-six. Total body burden 4.2 μg.

This patient is the wife of Patient 110. In the interval of 1922 to 1923 she took some "emanation water." In 1929 and subsequently, she was given radium chloride by her husband for an intermittent tachycardia. The total dosage reached approximately 400 μg. over a two to three year period.

Six years ago, she was told that she had "insufficient calcium in her back." At that time she had severe pain and wore a Taylor brace for two years. Four years ago, the patient caught her heel on a stairway, fell, and fractured her left femur, and was on crutches for fourteen weeks.

Determinations revealed a total body burden of 4.2 μg. The patient denied symptoms referable to "radium poisoning." Roentgenography revealed minimal lesions of bone resorption in the skull, thoracic vertebrae, humerus, and metatarsals, and moderate to markedly severe lesions in the ilium, acetabulum, radii, ulna, femur, tibia, fibula, and tarsals (Fig. 33 and 34).

Fig. 34. Patient 111. Body burden 4.2 μg. Necrosis with sequestration of the head of the radius and multiple areas of bone resorption in the shafts of the radius and ulna.

Patient 110. White male, physician, aged eighty-one. Total body burden 5.4 μg.

This patient, one of the physicians who was most active in the administration of radium in the Chicago area, first took radium water orally for six to eight weeks in 1922. Until 1929, he took radium intramuscularly in an intermittent manner, and estimates that over the years he has taken 700 to 800 μg. of radium. Determinations revealed a total body content of 5.4 μg. Roentgenography demonstrated minimal to moderately severe lesions attributable to radium in the skull, thoracic and lumbar vertebrae, ilium, acetabulum, humerus, radius, ulna, femur, tibia, fibula, tarsals, metatarsals, and the phalanges of the feet. The patient remains steadfast in his belief that the clinical administration of radium is an innocuous procedure and not attended by any late sequelae.

Patient 106. White male, aged fifty-three. Total body burden 5.6 μg.

This patient, the son of Patient 104, received "about 20 injections" of radium intramuscularly twenty-six years ago at the age of twenty-seven years for acne rosacea. Those were administered and prescribed by the same physician (Patient 110) who had treated his father.

In 1948, he noted a bone sequestrum about $\frac{3}{8}$ inch in diameter protruding from the left ramus of the mandible. This occurred six additional times, and all teeth have been extracted since the onset of the mandibular necrosis.

Physical examination revealed a hypertension of 230/130. The total body burden of radium was 5.6 μg. Roentgenography demonstrated minimal lesions in the ilium, acetabulum, humerus, and tibia, and moderately severe to severe lesions in the mandible, skull, thoracic vertebrae, radius, ulna, femur, and fibula.

Patient L. White female, aged forty-eight. Total body burden 6.7 μg.

This patient was admitted to the Elgin State Hospital in January, 1931, with a diagnosis of schizophrenia. During 1931, she was given in-

FIG. 35. Patient 103. Body burden 5.6 μg. Destruction of the head of the left femur and early changes in the opposite femur in a patient who was unaware that she had received radium.

travenously 10 μg. of radium each week to a total dose of 260 μg. The shafts of the femora and humeri had several 1 to 3 cm. areas of bone resorption. The heads of both femora demonstrated several areas of apparent increased density as did the wings of the ilium. The trabecular pattern of the pelvis and heads and necks of the femora were abnormal. The tibiae and fibulae demonstrated extensive areas of bone resorption that lent a streaked appearance. The metatarsals and phalanges also had several small linear areas of bone resorption.

PATIENT 103. White female, aged sixty-three. Total body burden 6.8 μg.

In July, 1951, the patient developed severe pain in her left hip, which rapidly increased in severity and limited her ability to walk. During the previous twenty-eight years, the patient suffered from intermittent episodes of joint pain, malaise, and fever, which, on various occasions, were thought to be due to brucellosis.

She consulted a physician in Tucson, Arizona, in 1947, because of pain in the joints and fever. Roentgenograms of the long bones and skull were made in the course of his study. Multiple areas of bone resorption were seen in these bones, and it was the impression of her physician that the patient was suffering from multiple myeloma, although further diagnostic procedures were not carried out.

The patient was seen in Chicago in June, 1952, by Dr. T. Coogan, who had also been the physician of Patient 112. He recognized the bone lesions as being typical of those seen in other patients in our series and referred Patient 103 to us.

The patient, formerly the wife of a physician, was completely unaware of having received radium, either orally or parenterally. However, in 1925 to 1926, she was hospitalized in Chicago because of her complaints of muscle and joint pain. During this hospitalization, she received intravenous injections of a medication, and it is known that the parenteral use of radium by certain members of this hospital's staff was not uncommon at this time.

The total body burden of radium in July, 1952, was estimated to be 6.8 μg. Complete skeletal roentgenography revealed severe destructive lesions typical of radium poisoning in the heads of the femur (Fig. 35), distal ends of both tibiae and fibulae, and lesser typical lesions in the other bones of the skeleton.

Arthroplasty for the relief of pain in the left hip was carried out by Dr. Hatcher of the University of Chicago Clinics in 1953, and the bone fragments were analyzed for radium content (Table III—paper three[47a] of this series). During 1953, extensive metabolic studies were done in the Argonne Cancer Research Hospital, and the excretion rates that were found have been summarized (Tables VI and VII, paper three[47a] of this series).

PATIENT K.B. White female, aged sixty-six. Total body burden 2 to 10 μg.

The patient received 12 injections of radium chloride (probably 120 μg.) intravenously from her physician in 1922 as a "tonic." The estimated body burden of radium based on urinary excretion rates in 1947 was 2.5 μg. and between 2 and 10 μg. by external gamma-ray determination. In 1944, the patient developed "arthritis," which gradually involved the right hip joint and both knees and, by 1946, was bedridden most of the time as a result of the joint changes. Roentgenographic examination at this time demonstrated changes in the long bones and pelvis that were characteristic of radium toxicity.

On April 13, 1947, she was admitted to the University of Chicago Clinics with the principal complaints of a defect in mental concentration and inability to speak correctly. There was a gross irregularity of the cardiac rhythm, and the blood pressure was 205/110 mm. Hg. The patient was digitalized and discharged as improved on May 19, 1947. The erythrocyte

count was low and the sternal marrow on April 18, 1947, was considered to be extremely atrophic. An incomplete skeletal roentgenographic survey during the hospitalization revealed marked aseptic necrosis of the head of the right femur (Fig. 36), moderate necrosis of the left astragalus, collapse of the vertebral bodies T-8 and T-12, fragmentation of the left patella, "mottling" of the head of the femur and "streaking" of the tibiae and fibulae.

Patient 104. White male, aged seventy-six. Total body burden 10.7 μg.

The patient, a physician and dentist, was treated intravenously about eighteen years ago with radium chloride by another physician (Patient 110) for vague "arthritic" pains in both shoulders. Over a period of nine months, the patient probably received about 700 to 800 μg. of radium intravenously.

Approximately two years after the injections of radium, the patient began to have difficulties with his jaw, and, in the ensuing year, lost all of his teeth and the anterior half of the mandible. About five years ago, he began to experience "arthritic" pain in the right hip joint, which progressed in severity and resulted in limitation of motion.

Examination revealed a total body burden of 10.7 μg. Roentgenography demonstrated slight to severe bone changes attributable to radium in the mandible, skull, thoracic and lumbar vertebrae, ilium and its acetabula, glenoid, humerus, radius, ulna, femur, tibia, fibula metatarsals, and phalanges of the feet. The patient developed a rapidly progressive aplastic anemia and died in 1952.

Fig. 36. Patient K. B. Body burden approximately 10 μg. Destruction of the head of the femur.

Patient 107. White male, aged sixty-nine. Total body content 13.9 μg.

The patient's physician (Patient 110) gave him "about 25 injections" of radium at weekly intervals twenty-six years ago. The total quantity that was injected is unknown. He was suffering from no particular illness at the time; the radium was administered by his physician to "pep him up."

During the past ten years, "arthritis" has affected the feet, knees, and right shoulder. In May, 1950, the patient suffered a right hemiplegia from which he made a satisfactory recovery.

The total body radium burden is 13.9 μg. Roentgenography revealed a paucity of lesions attributable to the presence of radium. Only minimal changes of bone resorption were noted in the thoracic vertebrae, radius, ulna, fibula, metatarsals, and the phalanges of the feet.

DISCUSSION

In the United States, there are hundreds, perhaps a thousand individuals, who were given radium salts parenterally or orally twenty or more years ago. For the most part, these persons are unaware that they carry radium deposits. Moreover, until recently, the medical profession has not realized that these patients exist. The long latent period between the time of administration of the radium and the appearance of symptoms has, in several instances, led to a delayed recognition of the correct etiology of the bone lesions.

Hematologically, the findings in these patients are neither striking nor diagnostic of radium poisoning (Table III). One patient (Patient 104) a seventy-eight year old male, died at the age of seventy-eight years of a rapidly progressive aplastic anemia. The usual hematologic criteria of radiation damage to the erythropoietic system, such as abnormalities of the total white blood cell count, and atypical appearance of the white blood cells, did not differentiate in this series between those individuals carrying a very small and a very large body burden of radium. Moderate anisocytosis and poikilocytosis of the red blood cells seemed to be constant findings

in those individuals carrying more than 2 μg. of radium.

An analysis of several thousand roentgenograms done on 38 individuals who received radium therapeutically and 6 individuals who had been employed as "radium" dial painters demonstrates a general correlation between the level of body burden of radium and the frequency and severity of osseous lesions.

A study, which includes measurements done by Miller and Rowland, that is reported in paper three[47a] of this series, indicates that the radium solution prepared by the United States Radium Corporation for intravenous use was not contaminated at the time of preparation with more than 0.66 per cent of mesothorium. The authors believe that it can be assumed with confidence that the individuals in this series who received radium only by the intravenous or intramuscular routes were not contaminated with significant quantities of mesothorium or radiothorium. It is possible that the oral preparations may have contained mesothorium since it is a fact that one of the proprietary preparations, "Radiothor," used in the 1920s, contained relatively large amounts of mesothorium, and was so advertised. It must also be assumed that the dial painters from Ottawa, Illinois, included in our roentgenographic analysis, may have ingested mesothorium and radiothorium in addition to the radium present in the luminous paints.

R. D. Evans* reports the presence of radiothorium that may have been present as a daughter of mesothorium or as the orginal contaminant along with radium in bone samples from Patient 112. This patient received the "radium" orally. Studies are now in progress by Stehney and his associates on bone specimens from several of our patients in an attempt to obtain further data on the relative importance of the presence of mesothorium and radiothorium in the induction of neoplasia and the production of osseous lesions.

A clinically recognizable and typical osseous lesion (in the dentin of the teeth) has been observed in Patient 120, who carries a body burden of approximately 0.15 μg. of "radium." Since he received this radium as a solution ingested orally, the possibility of contamination with mesothorium and radiothorium has not been exclused. The early age (8 years) at which the patient received the radioemitter may also be a factor of importance in the production of the lesion at such an extremely low level of total body radioactivity.

At body burdens of 0.4 μg. and greater, clinically recognizable and unique bone lesions are, with but 3 exceptions, present (Fig. 2). These lesions increase in number, extent of distribution throughout the skeleton, and in severity with increasing body burdens of radioactivity. A notable exception is Patient 107, who carried 13.9 μg. of radium and demonstrates only minor bone changes in the radius, ulna, and fibula. This unique exception to the general pattern will be the subject of further studies in an attempt to elucidate the factors that have mitigated against damage to the bone.

Sarcomas were seen in 4 patients included in this study and in 2 additional Ottawa dial painters observed but not included in this report. Patient 309, a radium dial painter from the Ottawa area, developed a sarcoma of the head of the radius in 1932. Following amputation of the arm, the patient has survived and, when examined in 1952, carried a body burden of 0.52 μg. of "radium." The bones of the arm, amputated in 1932, are available for assay. Patient 105 received radium salts parenterally and probably orally. A metastasizing osteogenic tumor arose in this patient whose body burden was 0.86 μg. The ingestion of a mesothorium-containing oral preparation cannot be excluded. Bone samples are not available for assay.

Patient 313, a radium dial painter from the Ottawa area, developed a fibrosarcoma

* Personal communication: "The specimen was a 5.9 g. (wet weight) piece of the femoral epiphysis . . . . The sample contained 2.3 ± 0.4 μμc. radiothorium and a radiothorium to radium ratio of 0.0024 ± 0.0005."

to the greater tuberosity of the right ischium in January, 1953, and died on July 22, 1954. In the 1926 to 1928 era, she tipped the dial painting brush with her lips. It is probable that the paint, at this time, contained radiothorium and/or mesothorium. Bone samples are available for assay.

Patient 112, who received radium salts orally, developed a sarcoma of the tarsal scaphoid. The presence of mesothorium or radiothorium in this oral preparation has not been excluded, and analysis of bone samples will be carried out.

No patient who received radium salts exclusively by the parenteral route (and thus probably received *only* radium) has developed a bone neoplasm attributable to the presence of radium in the skeleton. However, significant non-neoplastic lesions are present in those individuals containing as little as 0.4 μg. of what has been shown to be only radium salts, uncontaminated with more than 0.66 per cent mesothorium.

ADDENDUM

Since the preparation of this paper 2 additional patients have developed sarcomata.

Patient R. Total body burden 3.6 μg., has developed an osteogenic sarcoma of the proximal end of the right tibia. This is the first of the Elgin Hospital series of patients to develop a neoplasm and occurs in a patient who, it is believed, received only Ra^{226} intravenously.

Patient 103. Total body burden 6.8 μg., has developed a sarcoma of the first sacral vertebra. A laminectomy was performed by Dr. Charles W. Elkins of Tucson, Arizona. The tumor was found to be present in the epidural space, and histopathologically is a poorly differentiated fibrosarcoma.

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SPOT ROENTGENOGRAMS IN CHOLECYSTOGRAPHY

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IN AN appreciable number of routine cholecystograms, the diagnostic value of a contrast-filled gallbladder shadow is seriously impaired by superimposed skeletal structures, intestinal gas, or intestinal radiopaque material.

The additional procedures necessary to free the gallbladder shadow from these interfering densities are time consuming of both personnel and equipment, generally requiring multiple projections in a variety of patient positions.

The problem of extrinsic opaque shadows has been aggravated by the use of 3-(3-amino-2,4,6-triiodophenyl)-2-ethylpropanoic acid as a contrast medium. While providing a gratifyingly dense gallbladder shadow, this medium introduces an increased incidence of unabsorbed contrast material in the intestinal tract. Efficient utilization of this desirable density requires a technique which permits isolation of the gallbladder shadow from confusing intestinal tract shadows.

The obvious procedure for elimination of extrinsic shadows is roentgenoscopy with

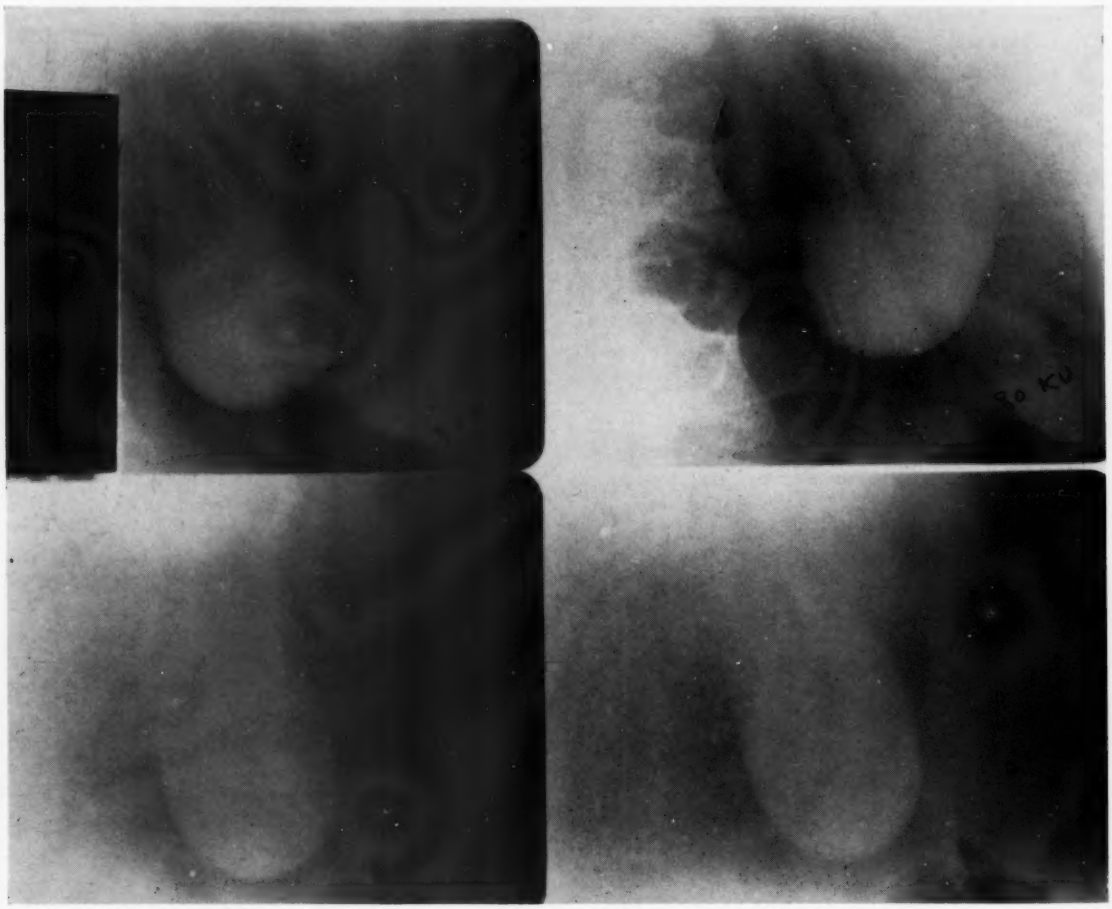

FIG. 1. Excellent spot roentgenograms are secured. Higher kilovoltage can eliminate the theoretical possibility of dense shadows obliterating small nonopaque stones.

FIG. 2. Case I. Small nonopaque stones confused by unabsorbed contrast medium in the colon.

FIG. 4. Case II. Poorly defined nonopaque stones confused by haustral gas shadows.

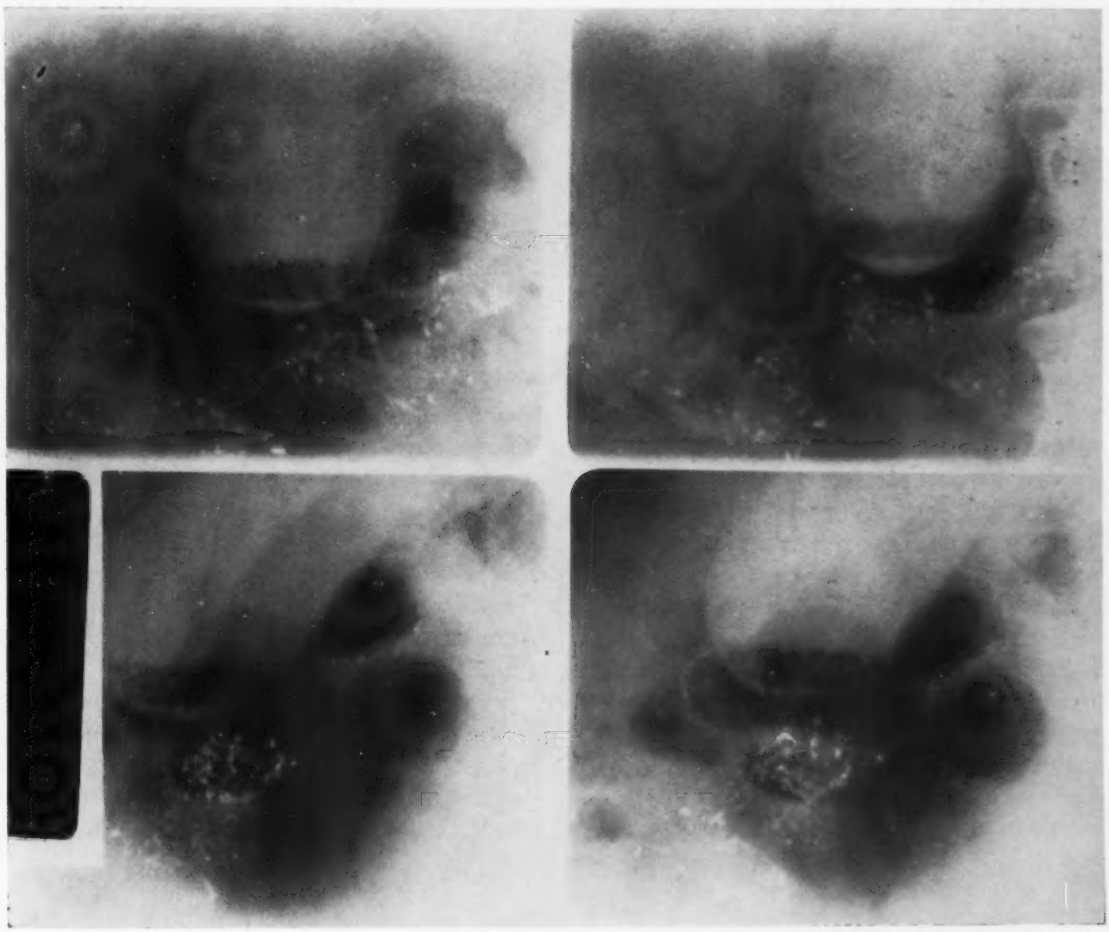

FIG. 3. Case I. Shows positive diagnosis secured by routine spot roentgenogram.

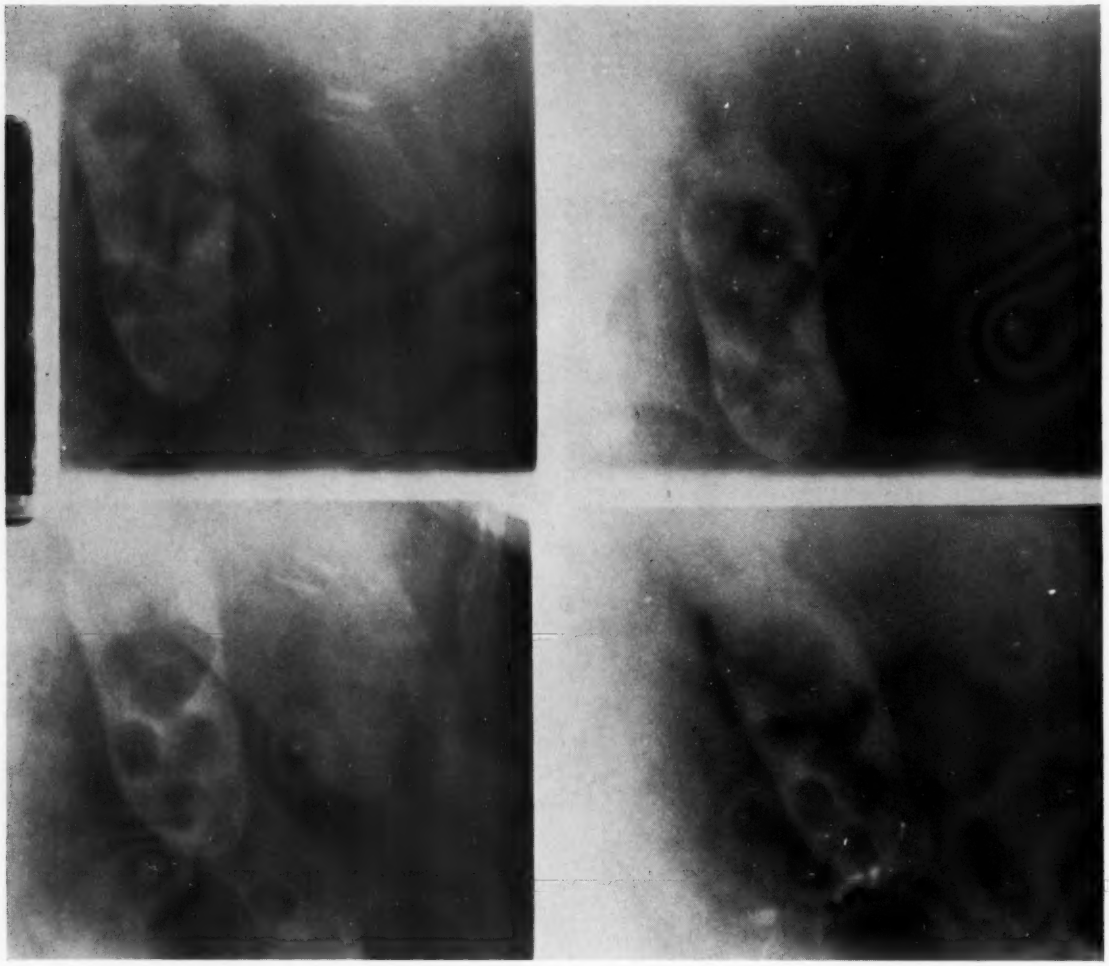

FIG. 5. Case II. Demonstrates positive diagnosis secured by routine spot roentgenogram.

the indicated compression and manipulation of the contrast medium and gas filled intestine. This procedure has been occasionally used in the past, but generally discarded as ineffective. However the availability of modern spot roentgenogram equipment with its remarkable roentgenographic potential has encouraged us to reconsider its use.

Routine roentgenoscopy and spot filming of cholecystography patients have been found practicable in our department. In the first 100 patients examined by this procedure, the gallbladder was visualized with sufficient clarity to permit effective compression and manipulation in 86 cases. Ten of the 14 not visualized at roentgenoscopy were "nonfunctioning," and the remaining 4 failures were due to patient obesity. The time required for this procedure averaged from two to three minutes. Four exposures were made on an 8 by 10 inch film, utilizing a rotating anode "undertable" tube with a focal spot of 1.5 mm. A grid was used in all cases. Exposures were at 100 ma., 80 (or less) kv., phototiming.

Following this, a slightly oblique prone 11 by 14 inch roentgenogram was made of the right abdomen to demonstrate possible neighboring pathology. In 80 of the 86 cases presenting a visible gallbladder shadow,

this procedure resulted in diagnostically adequate films. An examination following a fat meal was then made. In only 6 cases were further procedures needed.

The quality of the spot roentgenograms is surprisingly good in a range of 80 to 60 kv. (Fig. 1). The potential value of this procedure is apparent in that 4 of the 12 cases showing pathology (stones, polyp, calcium in the gallbladder wall) were diagnostically demonstrable *only* on the spot roentgenograms, and 24 of the normals could be cleared with certainty only with the aid of the spot roentgenograms.

Time studies by our chief technician indicate that this procedure results in a time saving of about 30 per cent in routine cholecystography. There has been a minimum of 25 per cent reduction in films used per person.

SUMMARY

1. Routine roentgenoscopy and spot films of cholecystography patients permit maximum exhibition of the gallbladder and produce roentgenograms of good quality with modern equipment.

2. A substantial saving is realized in time of personnel and equipment as well as film.

3. Diagnostic effectiveness is increased in a number of cases.

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Thirty-eighth Annual Meeting: Shamrock Hotel, Houston, Texas, April 9–11, 1956.

EDITORIALS

THE ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE Thirty-seventh Annual Meeting of the American Radium Society was held in Washington, D. C. on April 21, 22 and 23, 1955. The Shoreham Hotel and the attractive spring weather provided a wholesome atmosphere for the gathering. The total attendance was 374, including 122 members, 151 guests and 101 ladies. On the afternoon of the first day, the meeting was called to order by Dr. John E. Wirth who delivered the Presidential Address. He emphasized that the young radiotherapist of today has a better opportunity to make progress in the solution of the etiology and cure of cancer by having the advantage of improved training and instrumentation which only a few years ago were not available. The program continued with a spirited Symposium on Endometrial Cancer which was moderated by Dr. Franklin L. Payne of Philadelphia.

Following a social hour sponsored by the Radium Chemical Company of New York City, an informal dinner was held in the beautiful Terrace Room. Dr. Giacchino Failla introduced in a gracious manner Dr. Herbert M. Parker who delivered the Janeway Lecture. The title of his address was "The Radiological Sciences." Dr. Parker, who spoke in an unhesitating, witty and humorous vein, entertained the audience with important information relating to his work with the Atomic Energy Commission at Richland, Washington. He demonstrated with unique, cartoon-like slides the nature of his activities regarding pioneer methods of offering protection from radiation to humans, animals and plants in waterways, air and land in the neighborhood of an atomic reactor discharging its radioactive waste products into the river by sewer and into the air by smoke.

The morning session of the second day opened with two stimulating communications on radiation therapy of the urinary bladder, each one referring to a different type of modality. A lively discussion period ensued. Other papers in this session dealt with the use of radioactive gold in the treatment of malignant effusions, observations on the growth of human tumors, time-dose relationships and a new type of vaginal mold made of alginate dental impression material for the treatment of carcinoma of the cervix uteri. All of these papers were followed by an enthusiastic discussion period. The afternoon program contained a variety of material dealing with management of carcinoma of the base of the tongue, incidence of cataracts in irradiated patients, the effects of ACTH and cortisone on radiation pneumonitis, biological reorientation in radiation therapy, portable isotope roentgen-ray units and a comparator chamber for commercially available components. The incidence of cataracts and the need for biological reorientation in the study of cancer appeared to be the most stimulating and challenging addresses of this session.

On Friday evening a social hour for members and guests was held in the Foyer Terrace Banquet Room. At the Annual Banquet, Dr. Douglas Quick paid tribute in eloquent terms to Dr. Herbert M. Parker for his outstanding contributions in the field of radiation physics and presented him with the Janeway Medal. Following this ceremony, Dr. Wirth presented Honorary Membership Certificates to Dr. Manuel F. Madrazo of Mexico City, incoming President of the International

Congress of Radiology, Dr. Felix Leborgne of Montevideo, Uruguay, Dr. Oscar Soto of Lima, Peru, and Dr. Leonardo Guzman of Santiago, Chile.

The remainder of the scientific program on Saturday was devoted to a Symposium on the Clinical Diagnosis and Pathological Aspects of Various Benign Lesions of the Breast and Their Relationship to the Development of Carcinoma of the Breast. Dr. Cushman D. Haagensen of New York City was moderator of the panel in which four distinguished authorities on the subject participated.

The general enthusiasm of the members of the Society coupled with the pleasant atmosphere of the Shoreham Hotel made this meeting an outstanding success from both the scientific and the social aspects.

Harry Hauser, M.D.

STANLEY ALBERT WILSON
1897-1955

THE Salem Hospital in Salem, Massachusetts lost one of its most loyal workers and staunchest supporters, when Stanley Wilson died January 11, 1955, of coronary thrombosis. He had suffered his first coronary attack in November, 1954, and was convalescing satisfactorily, even planning a vacation in Florida, when he suffered the final attack. He had been the Roentgenologist at the Salem Hospital since 1937, succeeding Dr. Roy S. Perkins. In those eighteen years of devoted service to his hospital and to his patients he had built up an enviable record of accomplish-

ment. His work was always his first consideration and his colleagues came to depend upon him, not only for roentgenologic diagnosis, but also for clinical judgement and advice in many diverse problems. Having had seven years of general practice before going into radiology, he knew and appreciated the problems of his fellow practitioners. He was never too busy to talk over a case or a problem with one of his fellow physicians, or to encourage one of his own patients with kind words and sound advice. Except for the past few years he carried the entire burden of the roentgen department alone, and in addition had consultative responsibilities in other hospitals. Under his direction and guidance the Department of Roentgenology was developed from a small insignificant service employing only one technician to its present high efficiency, with two assistants, several technicians, an admirable residency-training program, and a place of importance and responsibility in the hospital and the community. In the past ten years he trained seven residents in radiology. All of them report the same characteristics in their Chief—kindness, consideration and understanding. His colleagues epitomized him as a man of charm and wit, bubbling with enthusiasm, with both the energy and ability to carry heavy responsibility. One of his greatest pleasures was to teach residents, and his contagious enthusiasm was an important factor in vitalizing the teaching aspects of the Salem Hospital internships.

Professionally he was thorough but fast in his work. His mind was keen and his brain worked in an orderly manner. He was tireless in his devotion to his duties and at all times honest, fair and reasonable in his conclusions as well as his criticisms. His abilities made his department one of the best in the towns around Boston, and his opinions earned the respect not only of his hospital staff but also of his radiological colleagues in the larger centers. He was President of the New England Roentgen Ray Society in 1949–1950, Secretary of the Medical Staff of the Salem Hospital for twelve years, 1941–1952, on the Staff Council from 1940 to 1954, and Chairman of the Hospital Planning Committee, all indicating the honor and trust given him, and the responsibilities gladly accepted by him.

His personality was best demonstrated in his therapy clinic where he cared for many patients with advanced cancer. There he was especially apt to reveal the kind heartedness, sympathy and understanding which characterize a good physician.

Stanley Wilson was born on March 19, 1897, in Brattleboro, Vermont, where he went through grade school and high school. His college work and his medical degree were both obtained at the University of Vermont, where he graduated with the M.D. degree in 1920. After an internship at the Mary Fletcher Hospital in Burlington, he went into general practice, first in Felchville, later in Richmond, Vermont. Becoming interested in roentgenology, he spent a year, 1930–1931, under Dr. David R. Bowen in the Department of Roentgenology of the Pennsylvania Hospital in Philadelphia, and also took post-graduate work at the Mayo Clinic, and at the Peter Bent Brigham Hospital in Boston. In 1932 he started his career in roentgenology in Burlington, where he was in charge of the roentgen-ray work at the Bishop deGoesbriand Hospital and the Fanny Allen Hospital. In 1935 he moved to Auburn, Maine, where for two years he was roentgenologist to the Central Maine General Hospital in Lewiston. In 1937 he accepted the position at Salem, Massachusetts, where he continued to work until his death.

Dr. Wilson was one of the first to become aware of the new and peculiar lung disease which turned up during World War II in persons working with fluorescent tubes. He worked long and hard against opposition and obstacles to identify this condition as an occupational hazard due to beryllium, and was among the first to publish accurate observations and conclusions on the disease now known as "berylliosis." He

followed these patients with personal attention and interest until the day of his death.

His hobbies and avocations were few, as his duties and responsibilities left him too little time for play. His chief delight in his later years was horseback riding, and at times he would have as many as four saddle horses or hunters in the stable at his farm in Hamilton, where he lived. His only other avocation was skiing, which he enjoyed with the same contagious enthusiasm as riding or jumping.

His family surviving him are his wife, Katherine Carpenter Wilson, whom he married in 1921, and two sons, Stanley, Jr. and Robert, the latter following his father's footsteps and now interning and specializing in radiology after graduating from Dartmouth College and Boston University Medical School. Dr. Wilson was also a 32nd degree Mason, a Shriner, and a member of the mounted patrol at Aleppo Temple in Boston. He belonged to the county, state and national medical societies, the American Roentgen Ray Society, the Radiological Society of North America, the New England Roentgen Ray Society (past-president) and was a Fellow of the American College of Radiology.

His record stands as an exemplary one of devotion to the ideals of furnishing the best possible radiological service to a large community hospital, and at the same time doing his utmost to improve the hospital for the benefit of patients and the community as a whole. His zeal was unflagging. His dedication to his goals was outstanding. His place in the hospital will, of course, be filled, but he can never be replaced in the minds or hearts of his patients, colleagues and friends. He will be remembered best as the composite of a skillful Roentgenologist, a wise counselor, a genial companion and a Christian gentleman.

Merrill C. Sosman, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS*

AMERICAN RADIUM SOCIETY

NEW OFFICERS

At the Thirty-seventh Annual Meeting of the American Radium Society held in Washington, D. C., April 21–23, 1955, the following officers were elected for the year 1955–1956: *President Elect*, Dr. Norman A. McCormick, Windsor, Ontario, Canada; *1st Vice President*, Dr. Herbert E. Schmitz, Chicago, Illinois; *2nd Vice President*, Dr. Manuel Riebeling, Guadalajara, Jal, Mexico; *Secretary*, Dr. Robert E. Fricke (re-elected), Rochester, Minnesota; *Treasurer*, Dr. Douglas J. Roberts (re-elected), Hartford, Connecticut. The *President* is Dr. Grant H. Beckstrand, Long Beach, California.

The Thirty-eighth Annual Meeting will be held at the Shamrock Hotel, Houston, Texas, April 9, 10 and 11, 1956.

ST. LOUIS SOCIETY OF RADIOLOGISTS

At a recent meeting of the St. Louis Society of Radiologists the following officers were elected to serve for the year 1955: *President*, Dr. Francis O. Trotter, Jr., St. Louis, Missouri; *Vice President*, Dr. Charles J. Nolan, St. Louis, Missouri; *Secretary-Treasurer*, Dr. Edwin C. Ernst, Jr., 3720 Washington Ave., St. Louis 8, Missouri.

SOUTH BAY RADIOLOGICAL SOCIETY

At a recent meeting of the South Bay Radiological Society Dr. Thomas N. Foster, 630 E. Santa Clara St., San Jose, California was elected *secretary*. This active group of radiologists meet on the second Wednesday of every month in the homes or offices of various members. Visitors are always welcome.

RADIATION THERAPY ALUMNI ASSOCIATION OF BELLEVUE HOSPITAL

A meeting of the Radiation Therapy Alumni Association of Bellevue Hospital was held on Thursday, May, 12, 1955, at which the annual Ira I. Kaplan Lecture was given by Dr. Gray H. Twombly, Professor of Gynecology, College of Medicine, New York University. Dr. Twombly's subject was "The Anatomy of the Female Pelvis in Relation to Cancer of the Cervix."

TWENTY-FIFTH ANNIVERSARY OF DR. ROBERT J. REEVES AS PROFESSOR OF RADIOLOGY CELEBRATED

The Twenty-fifth Anniversary of Dr. Robert J. Reeves as Professor of Radiology at the Medical School of Duke University, Durham, North Carolina, was celebrated on Friday, April 22, 1955. The Radiological Society of North Carolina invited Dr. Ross Golden, visiting Professor of Radiology, University of California at Los Angeles School of Medicine, and Emeritus Professor of Columbia University to address a meeting held at the Medical School of Duke University on Friday afternoon. Dr. Reeves took his residency in Radiology at the Massachusets General Hospital, Boston, under Dr. George W. Holmes. He was then associated with Dr. Ross Golden at the Presbyterian Hospital, New York, for four years. He went to Duke University as Professor of Radiology and Chairman of the Department of Radiology in 1930.

On Friday evening, April 22, Dr. Reeves was tendered a testimonial dinner, attended by many of his former residents and their wives. Dr. J. Stauffer Lehmann, Hahnemann Hospital, Philadelphia, was Master of Ceremonies. Dr. James P. Rousseau, President-Elect of the North Carolina State Medical Association and formerly Professor of Radiology at Bowman-Gray School of Medicine, Winston-Salem, North Carolina, was one of the speakers. Many gifts were presented to Dr. Reeves. A cocktail party at the home of Dr. Reeves on

* The list of Meetings of Radiological Societies appears in the May, 1955, issue of the JOURNAL, and will be published in the July number.

Saturday evening was a very pleasant occasion.

AMERICAN BOARD OF RADIOLOGY

The deadline for filing applications for the Fall 1955 examination by the American Board of Radiology is July 1, 1955. The examinations will be held the week of December 4, 1955 at the Palmer House in Chicago, Illinois. Those candidates who will complete their training by December 31, 1955 will be eligible to appear at this examination.

B. R. Kirklin, M.D.
Secretary-Treasurer
Kahler Hotel Bldg.
Rochester, Minnesota

DAVID ANDERSON-BERRY PRIZE (1956)

A David Anderson-Berry Silver-gilt Medal, together with a sum of money amounting to about £100, will be awarded in 1956 by the Royal Society of Edinburgh to the person who, in the opinion of the Council, has recently produced the best work on the therapeutic effect of roentgen rays on human diseases. Applications for this prize are invited. They may be based on both published and unpublished work and should be accompanied by copies of relevant papers. Applications must be in the hands of the General Secretary, Royal Society of Edinburgh, 22, 24 George Street, Edinburgh 2, Scotland, by March 31, 1956.

SCHERING AWARD

The tenth annual Schering Award competition is now underway. Subjects this year are: Current Concepts in the Management of Osteoporosis; Prevention and Treatment of Blood Transfusion Reactions; and Recent Trends in the Clinical Use of Adrenocortical Steroids. The aim of the Award is to encourage medical writing and exploration of current research, with the hope that students will contribute to the professional literature during their careers. Award winning papers themselves have been published and several of the new winners' papers are expected to be published this year. Inquiries for further information should be addressed to the Schering Corporation, Bloomfield, New Jersey.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Thomson, J. L. G. Thrombosis of major cerebral arteries. *Brit. J. Radiol.*, Oct., 1954, *27*, 553–564.

In 1951, Johnson and Walker reviewed the literature of carotid thrombosis and assembled 101 cases adding 6 cases of their own and discussing the important features at some length. Since then, several articles have appeared adding further cases to the literature. The present author reports a further series of 23 cases of carotid thrombosis and 7 of middle cerebral thrombosis and 1 case of posterior cerebral artery thrombosis. These cases were all seen in the Department of Radiology of the National Hospital, Queen Square, during the past four years.

Clinical Features. The youngest patient was twenty-four years old and the oldest seventy, with the average age about fifty years. Males predominated over females by $3\frac{1}{2}$ to 1. Left sided thrombosis was about twice as common as right sided and headache was present in 50 per cent of the cases. It was prominent in middle cerebral artery thrombosis from the start and was a dominant symptom in one case of posterior cerebral artery thrombosis.

Hemiplegia was present in 27 cases of varying severity.

Sensory disturbance occurred in 18 cases. Visual disturbance occurred in 13 cases, the commonest change being a homonymous field defect either partial or complete.

Loss of consciousness occurred in 3 cases each of internal carotid artery thrombosis and middle cerebral artery thrombosis.

Dysphasia occurred in 18 cases in one degree or another.

Some elevation of the blood pressure was present in 18 cases.

The type of onset was divided into two main groups: sudden hemiplegia, occasionally with loss of consciousness, occurred in half of the middle cerebral artery thromboses and in only a third of the internal carotid artery thromboses. The chronic onset occurred in the other thromboses of the internal carotid artery and in the remaining cases of middle cerebral artery thromboses.

The etiology of most of these cases was felt to be due to atheromatous degeneration of the internal carotid and middle cerebral arteries. Plaques of atheroma have been described as being frequently present at the lower end of the internal carotid artery, and it is theoretically possible for small thrombi, forming on them, to become dislodged and infarct small areas of brain, until a larger one blocks the carotid or middle cerebral artery. This point was illustrated in Case 18, at postmortem examination, which showed generalized atheroma of the internal carotid artery, middle cerebral infarction from a large embolus close to its origin, and older infarcted areas in the brain of about six weeks' standing.

Two cases were probably the result of embolism and in 2 other cases the patients were diabetics.

Arteriography. It is important to obtain roentgenograms which show both the stump of the block artery and the needle point in the neck, otherwise confusion could easily arise, as for example by puncture of the external carotid artery itself or by injection of most of the contrast material up the external carotid artery, should the needle be inserted just below its origin.

Analysis of this series shows that the block occurred at the following sites: 1 case showed a blocked common carotid artery, 16 cases showed a block at or within 3 cm. of the origin of the internal carotid artery with 14 of these exhibiting a definite stump. Two cases showed only an irregularity of the vessel wall at the expected site of origin of the internal carotid artery. Five cases showed a block in or about the carotid site. One case showed a grossly narrowed internal carotid artery but no absolute block.

A collateral circulation was outlined in three of these cases. The maxillary artery was hypertrophied to supply a vascular plexus in the orbit, from there the ophthalmic artery and the carotid siphon.

The middle cerebral artery block was usually conclusive. The posterior cerebral artery block showed a definitely reduced vascularity to the corresponding occipital pole with the posterior cerebral artery on that side almost completely blocked.

Discussion. The cases of internal carotid thrombosis and middle cerebral artery thrombosis were remarkably similar and less signs relating to anterior cerebral artery involvement were present. The fact that the anterior cerebral artery so often escapes may be explained by its normal anatomical connections which are well known to be variable. Both anterior cerebral arteries may be supplied from one carotid artery alone or both may be equally well filled from either side. The anterior communicating artery may also be large or small. Should the anterior cerebral artery be connected with one side only, it is likely to be involved in a thrombosis of the carotid artery. This state appears less common than the other in which the middle cerebral artery only is involved.

The clinical features showed close similarity, with the onset appearing to be commonly more rapid in middle cerebral artery thrombosis. Less than a third of the cases of carotid artery thrombosis had a sudden onset. The clinical picture also depended on the degree of collateral circulation available, either across the circle of Willis from the other side or through enlarged orbital anastomosis from the external carotid artery. The fact that so many carotid arteries are tied surgically as a therapeutic measure without signs developing suggest that there must be many symptomless carotid thromboses among apparently normal people.—*Arthur B. Smith, M.D.*

CAMPBELL, WILLIAM. The Treacher Collins syndrome. *Brit. J. Radiol.*, Nov., 1954, *27*, 639–641.

A syndrome of deformity of the face and skull was described by Treacher Collins in 1900. Straith and Lewis in 1949 published an account of a mother and 4 children, all of whom showed evidence of the syndrome. Roentgenologically, the outstanding features of the syndrome are:

(1) Agenesis of the malar bones.
  (a) Non-fusion of the zygomatic arches
  (b) Absence of the bodies of the malar bones
  (c) Ossification of each malar bone from two centers, with non-fusion of the frontal process.
(2) Agenesis of the palatine bones.
(3) Cleft palate.
(4) Agenesis of the mandible, unilateral and/or bilateral.

The author reports 2 cases of the syndrome, one a boy of eight, the other a woman of twenty-two. Neither patient gave a family history of any similar deformity. Each patient presented a typical appearance of "bird face," that was largely unilateral in the boy, but bilateral in the woman. The boy had an I.Q. above normal; the woman was of subnormal intelligence. The boy was considered a suitable subject for surgical treatment, and had had a prosthesis fitted to his palate, since the cleft in the palate was too wide for closure. He had no external auditory meati, but apparently normal labyrinthine and middle ear development; by surgery, a meatus was opened on each side. Mandibular surgery and reconstruction of the pinnae were contemplated at a future date.—*E. C. Davis, M.D.*

KARLSSON, K., ENGSTRÖM, A., and ENGSTRÖM, H. Microradiographic studies of the auditory ossicles (malleus and incus) and of the osseous labyrinth. *Acta radiol.*, Nov., 1954, *42*, 381–390.

The authors give a simplified theory for microradiography and detailed experimental techniques to demonstrate distribution of mineral salts in thin bone sections. Resolution better than 0.5 microns may be registered. The photographic emulsion is the main obstacle in obtaining higher resolution.

Microradiographic examination of thin ground sections from the malleus, incus and osseous labyrinth revealed a similar mineral pattern in a limited number of cases studied. It seems, however, that it may prove possible to describe the pattern of the distribution of mineral salts and its changes with age by a comparative study of a large number of cases in which all ages are represented. Such a study will form the basis for further investigation of otosclerotic processes.—*George A. Miller, M.D.*

NECK AND CHEST

McLEAN, MURIEL M. Chronic idiopathic hypoparathyroidism associated with moniliasis. *Arch. Dis. Childhood*, Oct., 1954, *29*, 419–421.

In 10 reported cases fungus infections have been found along with parathyroid deficiency. An additional case of such coincidence is reported. An eleven year old girl studied for idiopathic hypoparathyroidism was found to have monilial infection of the mouth, and ridging, thickening, and cracking of the finger and toenails. History indicated that the changes in the nails had begun approximately six years earlier. Typical grand mal seizures, presumably due to the hypoparathyroidism, had begun two years earlier. Although the nail changes may have been due to lack of calcium, the mouth lesions were most certainly due to chronic moniliasis.

The frequency with which coincidental monilial infection and idiopathic hypoparathyroidism have been reported indicates that it is more than a chance finding. It has not been reported in parathyroid deficiency following thyroidectomy. In some cases it has preceded the appearance of symptoms of hypoparathyroidism.

It seems unlikely that the fungus infection is responsible for depression of parathyroid activity, but there may be some factor which depresses the parathyroids and also encourages fungus infections.—*Lois Cowan Collins, M.D.*

SELDINGER, SVEN IVAR. Localization of parathyroid adenomata by arteriography. *Acta radiol.*, Nov., 1954, *42*, 353–366.

In recent years, hyperparathyroidism has been found to be more frequent than supposed. The radiologist may see striking changes brought about by disturbances in calcium metabolism, but has been of little help in localizing the parathyroid adenoma. It is often difficult or impossible to localize the enlarged gland at surgery.

Recently, it has proven possible to localize these tumors by contrast filling of the inferior thyroid artery with injections through a polyethylene catheter, the tip of which is placed in the subclavian artery, a little distal to the thyrocervical trunk. Displacement of the branches of the inferior thyroid artery, closely adjacent to and probably supplying the adenoma, should be looked for.

The author gives a careful description of the embryology, gross anatomy, blood supply and location of the normal and adenomatous parathyroid glands.

Parathyroid adenomas were localized roentgenologically in 4 out of 6 cases explored; 2 of these 4 adenomas had not been found at previous surgery. One complication is reported and discussed which was due to injection of an excessive amount of contrast medium into the vertebral artery.—*George A. Miller, M.D.*

Soutter, Lamar, Sniffen, Ronald C., and Robbins, Laurence L. A clinical survey of adenomas of the trachea and bronchus in a general hospital. *J. Thoracic Surg.*, Oct., 1954, *28*, 412–430.

This is a clinical study of the 56 cases of carcinoid adenomas and four cylindromas diagnosed at the Massachusetts General Hospital from 1909 to 1954.

In studying the carcinoid adenomas pathologically, it was found that 25 of the 30 encapsulated tumors showed microscopic evidence of penetration of the capsule. Metastases to adjacent lymph nodes occurred in three cases. The bulk of the tumor mass was usually outside the bronchus. Clinically, the most frequently encountered symptoms were cough, hemoptysis, and history of repeated episodes of pneumonia. Chest roentgenograms were available in 46 of the cases of carcinoid adenoma and atelectasis was demonstrated in 35 of the 46 studies. Bronchiectasis with and without collapse was present in 26 cases. In half of the cases the tumor mass was obscured by the inflammatory reaction in the adjacent lung. Calcification was demonstrated in 6 of the carcinoid adenomas roentgenologically. The extent of the surgical therapy was dictated by the location and extent of the tumor and amount of associated pulmonary damage secondary to sepsis. In the series, 15 pneumonectomies, 12 bilobectomies, 14 lobectomies and 2 segmental resections were done for carcinoid adenoma. One bronchotomy with removal of adenoma and 1 bronchoscopic removal of tumor were done. Thirty-three of 41 cases of carcinoid adenoma diagnosed before 1949 (73 per cent 5-year survival) are living. Both of the patients found to have metastases at surgery are living and well, one seven years and the other seventeen years after surgery. Fifteen patients in this series of carcinoid adenoma died. Ten were autopsied and only one of these, showed metastatic disease. This metastasis involved a single lymph node and apparently did not contribute to the patient's death. The clinical course of the remaining 5 patients, suggested that none died of metastatic disease.

The experience with cylindroma was small (4 cases). These tumors produced irregular infiltration with resulting constriction of long segments of the trachea and bronchi. While the cylindromas were slower in development and less lethal immediately than bronchogenic carcinomas, they were far more malignant in behavior than the carcinoids.—*Paul C. Olfelt, M.D.*

Ingelfinger, Franz J., Kramer, Philip, and Sanchez, Guillermo C. The gastroesophageal vestibule, its normal function and its role in cardiospasm and gastroesophageal reflux. *Am. J. M. Sc.*, Oct., 1954, *228*, 417–425.

The abnormal mechanism underlying cardiospasm and gastroesophageal reflux is poorly understood, partly because so little is known concerning the normal structure and function of the distal esophagus.

Anatomically Leriche divides the esophagus into the following areas: (1) the body of the esophagus extending from the upper esophageal orifice to the inferior esophageal sphincter (an inconstant constriction 2–3 cm. above the gastroesophageal junction); (2) the ampulla which comprises the terminal few centimeters immediately above the inferior esophageal sphincter; and (3) the gastroesophageal vestibule, the distal 2–3 cm. between the inferior esophageal sphincter and the gastroesophageal junction.

The esophagus can be similarly divided on the basis of its motor response to swallowing as evidenced by manometric studies. In the body of the esophagus there is an initial small abrupt generalized increase in intraluminal pressure when material is forced into it from the oropharynx. This is followed by the peristaltic pressure wave caused by a sharply defined segmental contraction which sweeps the contents down the esophagus ahead of it. The initial increase in pressure does not appear in the vestibule suggesting that this portion of the esophagus is normally closed at the onset of swallowing and the peristaltic wave ends before reaching this area.

Further evidence that the vestibule possesses characteristic and independent motility from the rest of the esophagus may be found by examining its function in patients with cardiospasm. In this condition the motor mechanism of the entire esophagus is deranged. The distal 2–4 cm. is contracted whereas the body is hypotonic and displays disorganized motility. The concept that this is due to vestibular dysfunction explains why the narrowed area always appears in the form of a segment, never a thin annular constriction, and supports Meltzer and Hurst's hypothesis of achalasia.

This concept can be further tested by response of the esophagus to cholinergic stimulation. In patients with cardiospasm mecholyl produced a tetanic contraction in the distal half of the body of the esophagus while the vestibule remained either unchanged or actually became dilated.

Anticholinergic stimulation in normal patients caused eradication of all vestiges of the peristaltic pressure wave with widening of the esophageal lumen, whereas the vestibule remained tightly closed.

Cardiospasm therefore appears to be a failure of vestibular relaxation or achalasia. A failure of the vestibule to remain contracted during swallowing may also occur and be the cause of gastroesophageal reflux.—*William C. MacCarty, Jr., M.D.*

Patterson, William H., and Fawcitt, John. Non-traumatic mediastinal emphysema in childhood. *Arch. Dis. Childhood*, Oct., 1954, *29*, 451–454.

The authors describe 4 instances of mediastinal emphysema in infancy and childhood which was sec-

ondary to recognized pulmonary disease. The cases presented were coincident with pertussis following measles by three weeks, an acute asthmatic attack, bronchopneumonia and probable tracheitis with collapse of the middle lobe. The clinical symptoms include sudden onset of chest pain and dyspnea with an absence of constitutional symptoms.

The precursor of nontraumatic mediastinal emphysema is almost invariably pulmonary interstitial emphysema. The authors stress the value of roentgenograms in the oblique and lateral projections in detecting small amounts of air in the anterior and posterior mediastinum. The air is generally shown in the form of streaky shadows which accentuate the outlines of the organs and vessels of the mediastinum. With increasing amounts of air it may extend into the neck or produce a pneumothorax.

The authors quote Macklin in the theory of etiology. Hyperinflation of the alveoli bordering the blood vessels tends to build up an intra-alveolar pressure greater than that within these vessels, causing a rupture of the alveolar bases and escape of air into the perivascular sheaths. These tiny bubbles of air pass down the sheaths towards the hilus, and, tending to coalesce, form blebs of air in the hilar region whence they burst into the mediastinum. There are also extensions of air into the contiguous connective tissue formations in the region of the hila. This mechanism not only causes a degree of vascular stasis due to the air in the perivascular sheaths occluding the vessels, but reduced respiratory excursion by "splinting" the lung tissue.

The air may follow three possible courses when it reaches the mediastinum: (1) remain in the mediastinum, (2) produce a pneumothorax, or (3) track upward into the neck, trunk and thorax.

The condition may be associated with any lesion causing localized atelectatic changes in the lungs which could be surrounded by an area of hyperinflated lung tissue.—*James F. Martin, M.D.*

Wyman, Stanley M. Angiocardiography; a guide to mediastinal exploration. *New England J. Med.*, Oct. 28, 1954, *251*, 723–729.

The use of angiocardiography as an ancillary method of evaluation of abnormal mediastinal shadows is discussed. Nine cases are presented: (1) dilated pulmonary trunk, (2) a mass of matted lymph nodes, (3) absence of the left pulmonary artery, (4) anomalous right subclavian artery with dilatation of its proximal portion, or of some form of aortic diverticulum, (5) a mass close to the left subclavian artery and near the aortic arch—it did not opacify, (6) coarctation of the aorta with poststenotic dilatation, (7) persistent left superior vena cava and dilated right superior vena cava, giving Snellen's "figure of 8" configuration (total anomalous venous connection), (8) an "enlarged thymus" in a five year old child, and (9) an aberrant right subclavian artery giving the appearance of a para-esophageal mass in a sixty-two year old man with a carcinoma of the esophagus.

The author concludes that the method is undoubtedly of enormous value, both positive and negative; it strengthens the case for mediastinal exploration in some patients and in others establishes the fact that thoracotomy is unnecessary and possibly dangerous.—*André Bruwer, M.B., Ch.B.*

Batt, Harold D., Allen, John M., Treder, Frederick H., and Shapiro, Robert. Photofluorographic detection of cardiovascular disease in a general hospital. *New England J. Med.*, Dec. 2, 1954, *251*, 934–937.

This is a study of the detection of cardiovascular disease on 70 mm. photofluorograms made on all adult patients admitted to the Hospital of St. Raphael, a general hospital, during the year 1953. In addition to the routine posteroanterior film, lateral films were taken on all patients except the very obese and the prenatal group. Almost every film was interpreted by a third year resident and all films were finally examined by a senior radiologist particularly interested in cardiovascular disease. Patients with suspicious cardiovascular silhouettes were examined further by means of urinalysis, complete blood count, serologic test for syphilis and, whenever possible, an electrocardiogram and clinical evaluation by a resident in cardiology. Roentgenologic study consisted of teleroentgenogram or cardiac fluoroscopic examination or both.

Of the total of 6,439 adults surveyed, 709 were suspected of cardiovascular disease. In the follow-up studies of 595 patients, 497 (83 per cent) were shown to have cardiovascular disease. Of the 595 patients, 416 were evaluated for prior knowledge of cardiovascular disease. Forty-seven per cent (196) were unaware of such disease.

The surgical group of 3,305 adults showed 512 (16 per cent) with abnormal cardiovascular silhouettes. Of 437 patients studied, 368 (84 per cent) were shown to have cardiovascular disease.

The medical group consisted of 477 patients. Of these, 156 (32 per cent) had abnormal photofluorograms. Of 134 followed, 121 (90 per cent) had cardiovascular disease.

There were 2,657 women in the young obstetric group. Forty-one (1.5 per cent) had abnormal cardiovascular silhouettes. Of these, 24 were followed and 8 (33 per cent) were proved to have cardiovascular disease.

The lateral photofluorogram helped to increase the accuracy of the interpretation on the posteroanterior film, but no cardiovascular abnormalities were detected solely on the lateral film.—*J. N. Ané, M.D.*

Steiner, Robert E. The radiology of respiratory distress in the new born. *Brit. J. Radiol.*, Sept., 1954, *27*, 491–499.

The delicacy of the newborn, even when premature, is not such that roentgen examination of the chest should be feared whenever respiratory distress is present. If necessary, a mobile unit can be employed. In fact, chest roentgenograms should be obtained as soon as respiratory embarrassment is recognized. Often serial studies are indicated. The roentgen examination usually leads to earlier diagnosis and therefore to prompter, more effective treatment of many conditions such as atelectasis, pneumonia, pneumothorax, diaphragmatic hernia, and hyaline membrane syndrome.

Accurate roentgenography and reliable interpretation are entirely possible in the newborn.—*George Cooper, Jr., M.D.*

Krabbenhoft, Kenneth L., and Evans, William A., Jr. Some pulmonary changes associated with intracardiac septal defects in infancy. *Radiology*, Oct., 1954, *63*, 498–507.

In studying a collection of chest roentgenograms of infants and children dying from congenital cardiovascular malformations, and then subjected to careful autopsy, the authors found a high incidence of atelectasis and emphysema in the lungs of those with interatrial septal defects. This observation proved valid when the group with intracardiac septal defects was compared with a group of other cardiovascular anomalies. It was felt that this should be of some diagnostic and prognostic significance.

Specifically, the lung changes in the group with intracardiac, and especially interatrial septal defects, showed the atelectasis tending to involve the upper lobes, while emphysematous changes occurred predominantly in the lower lobes.

In an attempt to determine the etiology of the high incidence of these pulmonary changes, several hypotheses are considered: (1) That these pulmonary manifestations represent a basic structural abnormality of the bronchi or lungs which predisposes to respiratory infection. (2) That the disturbance of the pulmonary circulation associated with the septal defect produces, an inadequate pulmonary function which predisposes to infection, emphysema, and atelectasis. (3) That these infants are otherwise peculiarly susceptible to respiratory infection. The evidence at hand does not decisively support any of these contentions, but the authors are inclined to favor the first hypothesis, in that there is often an abnormal anatomical structure of the lung which predisposes to persistent and recurrent respiratory infection, emphysema, and atelectasis in patients with interatrial septal defect and to a lesser extent with other types of intracardiac defects.

Several interesting cases in each category of intracardiac septal defects are presented to illustrate these findings and the authors' viewpoints.—*Michael J. Healy, M.D.*

Mitchell, Fred N. Pulmonary arteriovenous telangiectasia. *South. M. J.*, Dec., 1954, *47*, 1156–1161.

The author reports 7 cases of pulmonary arteriovenous telangiectasia to bring the total number of reported cases to approximately 87. Of these, 33 had a family history compatible with familial hemorrhagic telangiectasia.

In 1 of the cases of the author's series a similar lesion occurred in a nephew. This represents the first reported case occurring outside of an immediate family.

It is important, also, that in 1 case of this group the apical posterior segment of the left upper lobe was involved. Since Watkins and Harper had noted that pulmonary arteriovenous telangiectasias had never been reported in the apices of the lungs, this case is noteworthy. It is plausible to assume that the lesion may occur in any portion of the lung.

In the study of these cases the author has stressed the importance of family history, fluoroscopy, the Valsalva and Muller maneuvers and angiograms.—*J. N. Ané, M.D.*

Hermel, M. B., and Gershon-Cohen, J. Healing mechanisms of tuberculous cavities. *Radiology*, Oct., 1954, *63*, 544–549.

Closure of cavities is the primary aim in all forms of medical and surgical therapy in pulmonary tuberculosis.

Tuberculous cavities heal by two general processes, open or closed, the particular designation depending upon the status of the draining bronchus. Open healing is rare, while closed healing is common. Closed healing occurs either by atelectasis and scarring of the cavity or by inspissation. A cavity which blocks intermittently sometimes increases in size and rarely eventuates in healing. It is frequently the cause of extension of the disease with contralateral spread at times of released tension.

Roentgenograms illustrating the various forms of cavities with follow-up studies of 4 cases are reproduced.—*E. E. Seedorf, M.D.*

Abdomen

Riker, William L. Congenital diaphragmatic hernia. *A.M.A. Arch. Surg.*, Sept., 1954, *69*, 291–308.

The author presents a very interesting discussion of 33 cases of congenital diaphragmatic hernias. These are classified into (1) short esophagus and true hiatus hernia; (2) hernia through the foramen of Morgagni; (3) hernia through the foramen of Bochdalek; (4) eventration of the diaphragm; and (5) congenital absence of the diaphragm. Of these cases 31 have undergone surgical repair. In the general analysis of the group the author emphasizes that the patients with congenital diaphragmatic hernias

under one year of age, especially under one week, require emergency operative repair as the symptoms are usually severe. In those patients where the hernia is discovered after one year of life the symptoms are milder or absent and the surgical repair is on an elective basis.

In the 31 surgically treated cases there were six deaths. Four of these were operative deaths and the other two patients died later from other congenital anomalies. All of the surviving patients have done well with only one patient developing a mild recurrence of the esophageal hiatus hernia which was not of sufficient degree to require further treatment.

Congenitally short esophagus was present in only one instance. In these cases mucosal ulceration usually causes stricture at the esophageal junction which for the most part is successfully treated by dilatation. The author's case was that of an infant seven days old and the congenital constriction required operative intervention at that time. The esophageal hiatal opening was brought to the level of the gastroesophageal junction and sutured into place. The patient died one month later from a congenital hydronephrosis and at post-mortem examination the repair was still intact.

True esophageal hiatus hernia is usually an incidental finding in infancy. In the patients without symptoms operative repair is not urgent although the author feels that the simple operation of inversion and imbrication of the sac with snugging of the esophageal hiatus is sufficient in infancy while it would not be so in adults.

The majority of the cases in this series had herniation through the foramen of Bochdalek. This pleuroperitoneal canal is the last area to close in the embryological development of the body cavities, the closure being normally completed between the eighth and ninth week of fetal life. If there is too rapid return of the intestines to the abdominal cavity or a tardy closure of the diaphragm, intestines may find a way into the pleural cavity through the uncompleted diaphragm. These patients often are acutely ill since cyanosis and dyspnea develop rapidly as the bowel fills with gas and food. Roentgenograms readily establish the diagnosis and a lateral view of the chest helps localize the defect of the diaphragm. The author mentions the difficulties in closing the defect due to obtaining enough tissue to bridge the gap as well as being unable to replace the intestines in the abdominal cavity. In some cases only the skin could be closed, with a secondary closure of the peritoneum and fascia ten to fourteen days later. The sole case of hernia through the foramen of Morgagni produced a similar clinical picture to that seen in the posterior portion of the diaphragm with relatively the same surgical procedure for repair.

In eventration of the diaphragm symptoms are usually absent. Diagnosis is made primarily by roentgen means. Unless the eventration is marked and symptoms are present surgical repair is not recommended. The author has no instance of complete absence of the diaphragm in this series of cases.

The article is well illustrated with roentgenographic reproductions of the various types of diaphragmatic hernias.—*Thomas D. Allison, M.D.*

Even, Roger, Sors, Christian, and Codis, René. Les hernies diaphragmatiques rétro-costo-xiphoïdiennes. (Retro-costo-xiphoid diaphragmatic hernias.) *Presse méd.*, Sept. 29, 1954, *62*, 1302–1304.

These hernias constitute 3–5 per cent of all diaphragmatic hernias. The herniation occurs through the space of Larcy or immediately near this area. (In this country it is usually referred to as the retrosternal space or foramen of Morgagni.) They are observed in all age groups, and occur more frequently in men.

The hernias are simple or mixed. The mixed forms are gastrointestinal, epiplointestinal or diverse. Some are latent with no symptoms. Others produce symptoms which may vary with the contents of the hernia. The gastric and intestinal forms present digestive disturbance and radiolucent areas in the hernia. Those containing epiploon or liver may lead to respiratory symptoms and exhibit a roentgen opacity.

Oral barium studies and pneumoperitoneum are of aid in differential diagnosis. Localized hydropneumothorax, pleuropericardial cysts, deformities of the diaphragm and teratomas should be considered in the differential diagnosis.—*Charles M. Nice, Jr., M.D.*

Cosco Montaldo, Homero. Quiste hidático del higado; contribución al diagnostico precoz. (Hydatid cyst of the liver; contribution to its early diagnosis.) *An. Fac. de med. de Montevideo*, May–June, 1954, *39*, 249–266.

Although in Uruguay a semispherical elevation of the right dome of the diaphragm (image "en brioche") is highly suggestive of hepatic hydatid cyst, other conditions as true hernia of the liver, omental hernia of Larrey's cleft, diaphragmatic irregularities due to postoperative adhesions, and mediastinal tumors can produce the same roentgenological image. The arc appearance, observed mostly in young, uncomplicated, generally hyaline, and univesicular cysts is, according to the author's experience, a sure indication of hydatid cyst. It consists of a faint, at times almost invisible line as if traced with a fine pencil, it measures from 1–1.5 mm. in thickness, is located in the substance of the liver and is concave upwards. It is detectable in anteroposterior roentgenograms of the liver and especially in cholecystograms and at times also in lateral views. The line represents the pericystic membrane and has to be differentiated from the outline of a calcified cyst, which is easily identified for its irregular, thick, and broken appearance.—*F. Comas, M.D.*

GAEBEL, E., and TESCHENDORF, W. Über Fortschritte in der röntgenologischen Darstellung der Gallenblase. (Progress in the roentgen visualization of the gallbladder.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Sept., 1954, *81*, 296–313.

After briefly mentioning the newer tri-iodo compounds for peroral cholecystography (telepaque and teridax), the authors describe their experience with a new compound for intravenous examination of the biliary tract introduced by Schering in Germany under the trade name of biligrafin and containing 6 iodine atoms per molecule. This substance is excreted rapidly and about two hours after injection a good gallbladder opacification is observed. There are few side reactions and no iodism is noted.

It is used in 20 per cent solution and the usual dose is 40 cc. A 40 per cent and 52 per cent solution have been recently developed but are not yet on the market. These two were used in doses of 10–20 cc. and give a denser opacification. Side effects consisting of nausea, vomiting and diarrhea are more frequent.

In patients with markedly impaired excretory function of the liver, the opaque medium is excreted mainly by the kidneys. No permanent liver damage has been observed in these patients.

Absence of the opaque medium in the biliary tract is found in patients with severely impaired liver function and in fistulae between the biliary tract and the bowel.

The differential diagnosis between obstructive and hepato-cellular jaundice can be made sometimes by this method. Complete obstruction, mainly by tumors, will cause markedly increased pressure of the excreted bile with nonvisualization of the biliary tract. Jaundice without too severe liver damage will give a positive intravenous cholangiogram.

Visualization of the ducts only is found, apart from cholecystectomized patients, in complete obstruction of the cystic duct and in contracted gallbladders with obliteration of the lumen as well as in congenital absence of the organ.

Stratification of bile of various densities in the gallbladder can be mistaken for nonopaque concrements. The degree of opacification of the gallbladder does not depend on the concentrating ability of the mucosa and consequently a diseased organ can be well opacified.

The authors give the following indications for examination with biligrafin: (1) Absence of opacification by the oral method. In these patients concrements in the gallbladder and the ducts can be demonstrated. Dilatation of the ducts and kinking due to elongation of the common duct is often associated with stones and persists for some time after their removal. (2) Patients with postcholecystectomy syndromes. These showed the following findings: (a) obstruction by residual or newly formed stones; (b) dilatation of the remaining stump of the cystic duct with or without concrements; (c) retrograde filling of the duodenal bulb by the opaque medium which might be mistaken for a dilated cystic duct.

Adhesions are difficult to demonstrate; examination of the gastrointestinal tract and pneumoperitoneum with lateral roentgenograms with the patient supine are useful.

Patients after cholecystectomy, examined by this method, frequently show slight to marked dilatation of the ducts. This in absence of obstruction is usually due to some unrelated disease as gastric and duodenal ulcers, pancreatitis, appendicitis or so-called odditis. In this condition a dilated common duct with a narrow comma-shaped lower end is found which is ascribed to a spasm of Oddi's sphincter, secondary as a rule to pancreatic disease. A barium meal examination will often demonstrate a widened duodenal loop with Fosberg's sign.

This article gives a well illustrated summary of the experiences based on over 250 intravenous cholangiographies performed with 40 per cent solution of biligrafin. An extensive list of references is added.—*Karol E. Matzinger, M.D.*

SEEDORF, E. E., POWELL, W. N., and DYSART, D. N. A comparison of five gallbladder media. *South. M. J.*, Sept., 1954, *47*, 809–813.

The authors review the development of the roentgen diagnosis of gallbladder disease, citing Beck's discovery of the shadow of a gallstone on a plain roentgenogram in 1900, and the well known work of Graham and Cole. They review further the historical sequence together with a brief study of the five contrast media used in cholecystography, *i.e.*, tetraiodophthalein, monophen, priodax, telepaque and teridax. One hundred patients were studied after the use of each of these media, excepting the tetraiodophthalein, with which only a few cases were examined because of the very small supply that could be obtained.

The study includes a comparison of these media as used in cholecystography, the incidence of side reactions, development of pseudoalbuminuria and an evaluation of the quality of the gallbladder shadow on a roentgenogram.

The authors stress the high degree of accuracy that has been obtained in the diagnosis of gallbladder disease. In the case of telepaque and teridax, they are inclined to attach little or no diagnostic value to the visualization of the bile ducts. Furthermore, they emphasize the fact that as small a dose as 2 grams of telepaque produced as dense a shadow as larger doses of the other media especially teridax, with a dose of 3.75 grams, although both are triodenated.

In their conclusion, they consider that the employment of any of the available cholescystographic media permits a high degree of diagnostic accuracy.

Three charts and reproductions of five roentgenograms accompany the article.—*William H. Shehadi, M.D.*

MOSCA, LIDIO G. La colecistocolangiografía por via bucal con Telepaque. (Oral cholecystocholangiography with telepaque.) *Prensa méd. argent.*, Oct. 22, 1954, *41*, 3087–3091.

The oral administration of telepaque gives a much more opaque shadow of the gallbladder than has been obtained with contrast agents previously used. At times opacification is sufficient as early as six hours after ingestion of the telepaque. The maximum opacity is reached by twelve hours and opacification is maintained longer than with agents formerly used. In addition, there are a few patients in whom visualization of the gallbladder is obtained by telepaque when previous agents yielded no shadow. When using telepaque the double-dose technique offers no advantage.

Patients tolerate telepaque better than other contrast agents. About 10 per cent complain of nausea and 15 per cent have diarrhea. Some of the opaque substance is not absorbed and is seen scattered through the colon, but this does not interfere with visualization of the gallbladder.

Opacification of the cystic and common ducts may be obtained in 82 per cent of patients if serial films are taken at 8, 12, 15, 20 and 30 minutes after a fatty meal.—*Charles M. Nice, Jr., M.D.*

ALMASQUÉ DEDEU, RICARDO, and BRAIER, LEONARDO OSCAR. Diagnostico y tratamiento de las discinesias biliares. (Diagnosis and treatment of the bilary dyskinesias.) *Prensa méd. argent.*, Oct. 29, 1954, *41*, 3151–3155.

A careful clinical study is necessary for diagnosis of the type of biliary dyskinesia which may be present before therapy is instituted. Cholecystography, duodenal drainage, roentgenologic examination of the digestive tract, examination of feces for parasites, studies of the neurovegetative system and biliary manometric studies during surgery may all be of aid in the complete analysis.

In hypotonia of the biliary tract the ideal treatment consists of stimulating function of the gallbladder. This condition may be accompanied by hypochlorhydria, gastroduodenitis, or constipation, in which case appropriate therapy must be given. In hypertonia of the sphincter of Oddi an antispasmodic is indicated. Novocaine, nitrites, and vagolytic substances are included in this group of medicinal agents. Sympathomimetic agents such as benzedrine, anesthesia of the splanchnic nerves and roentgentherapy may also be of value. Therapeutic duodenal drainage may be of value in postcholecystectomy hypertonia.

Stenosis of the cystic duct may also cause dyskinesia, and this calls for surgical intervention. When cholecystectomy is indicated, biliary manometric studies are also performed. If these are normal, cholecystectomy is the only procedure performed. If manometry indicates hypertonia, which is relieved by vagal infiltration, the vagus nerves are sectioned near the lesser curvature of the middle third of the stomach. If monometry indicates hypotonia, preganglionic splanchnicectomy on the right is performed. If manometry is normal and cholangiography indicates pancreatic reflux, without macroscopic pancreatic disease, left splanchnicectomy is indicated to suppress pain and improve pancreatic circulation.—*Charles M. Nice, Jr., M.D.*

ALMASQUÉ DEDEU, RICARDO, and BRAIER, LEONARDO OSCAR. Colangiografía intravenosa; diez casos operados. (Intravenous cholangiography; ten operated cases.) *Prensa méd. argent.*, Nov. 19, 1954, *41*, 3372–3374.

Intravenous cholangiography is performed with biligrafin, which contains 64 per cent iodine per molecule, and permits opacification of the biliary ducts without previous concentration in the gallbladder. Ten patients underwent intravenous cholangiography before surgery. In one patient a stone in the common duct was diagnosed, but the diangosis was not proved at surgery. In another a stone was present in the common duct at surgery which had not been visualized. In another patient a long cystic stump was visualized but removal of the stump did not ameliorate the patient's symptoms. The cystic stump was not visualized in one patient with a xanthogranuloma of the stump, nor in another patient with neo-vesicle containing a stone.

Intravenous cholangiography proved of value in 3 patients. In one a nonfunctioning gallbladder with normal ducts was diagnosed and confirmed at surgery. In another, the magnitude of a surgical lesion which the patient had was depicted. In a third patient an organic lesion of the papilla was demonstrated.

It is concluded that intravenous cholangiography is only part of the clinical study and further technical and interpretative improvements are desirable.—*Charles M. Nice, Jr., M.D.*

ESCOSTEGUY, GERALDO. Colangiografia intrahepática transoperatória (nota preliminar). (Operative intrahepatic cholangiography.) *Rev. med. Rio Grande do Sul*, July–Aug., 1954, *10*, 371–377.

The author has modified the technique of operative cholangiography by clamping the distal end of the common duct previous to the injection of the contrast medium. With this method the intrahepatic ducts are better visualized while the patency and functioning of the extrahepatic bile ducts can be studied in roentgenograms taken after removal of the clamp. It is the author's impression that this

method lends itself also to study of the pharmacological tests of the bile duct function.

Thirteen very good cholangiograms are reproduced in the paper.—*F. Comas, M.D.*

Sutton, David, and Tillett, John V. Intravenous cholecystography and cholangiography: clinical trials with a new medium (biligrafin). *Brit. J. Radiol.*, Oct., 1954, *27*, 575–581.

The authors review the development and progress made in the visualization of the biliary tract by oral media, since the initial work of Graham and Cole. They report on a new intravenously injected medium known as biligrafin.

One hundred and twenty patients were examined with biligrafin and were compared with 100 cases examined with telepaque.

The authors confirm the reports of German investigators who did the original work of biligrafin. They feel that the chief advantages of this new medium lie in the fact that (1) the intravenous route obviates the possible uncertainties of absorption of contrast media administered by mouth, (2) there is little or no toxicity, (3) excretion by the liver is rapid and is sufficiently concentrated to bring about adequate cholecystography and cholangiography, (4) cholangiography is possible after the gallbladder has been removed.

The bile ducts are visualized as early as fifteen minutes and the gallbladder is visualized in about two hours. The gallbladder has been demonstrated in a higher percentage with biligrafin than with telepaque, and the bile ducts have been more uniformly outlined.

In jaundice, the biliary tract was not visualized. In this respect, biligrafin offered no advantage over other orally administered contrast media.

While the authors feel that biligrafin presents greater diagnostic accuracy, they do not believe that its intravenous use will replace the routinely employed oral contrast media, mostly because of the cost and the time involved, as well as the possible complications of intravenous injection.

Twelve reproductions of illustrative roentgenograms and two tables are presented.—*William H. Shehadi, M.D.*

Mosca, Lidio G., Zorrilla, Juan I., and Montángero, N. R. La colangiografía por la vía intravenosa; caracteristicas, técnicas, indicaciones y resultados. (Intravenous cholangiography; characteristics, technics, indications and results.) *Prensa méd. argent.*, Sept. 17, 1954, *41*, 2719–2736.

Occasionally the cystic and common bile ducts are visualized following oral administration of telepaque if the concentrating and contracting powers of the gallbladder are normal. Intravenous administration of biligrafin (cholographin brand in U.S.A.) permits roentgen visualization of the extra-hepatic ducts in a higher percentage of cases, even when the gallbladder is absent. The iodine content of the latter is 64.32 per cent. Apparently concentration of the opaque substance occurs in the liver. Maximum concentration occurs in ten minutes. The LD50 is cited as 3.4 grams per kilogram body weight, viz., it is ten times less toxic than oral cholecystographic preparations. Two series totaling 1,500 cases are quoted as showing no serious complication following clinical use of biligrafin. Immediate reaction may consist of general discomfort, vomiting, redness, diaphoresis and other mild symptoms. In one case circulatory collapse occurred two hours after injection. This disappeared in twelve hours.

About 90 per cent of the opaque substance is excreted via the intestinal tract, and 10 per cent is excreted by the kidneys. This percentage is almost reversed with hepatic dysfunction.

The fasting patient is prepared with enemas and pitressin. The quantity of contrast agent recommended by the manufacturer is 20 cc. in those patients with an intact gallbladder, and 40 cc. in those in whom the gallbladder is absent. Injection of morphine to contract the sphincter of Oddi has not resulted in more intense opacification. The injection is performed slowly at about the rate of 20 cc. per three minutes. Low kilovoltage and high milliamperage are utilized in taking films.

The hepatic ducts begin to opacify ten minutes after the injection. The cystic and common ducts are opacified slightly later, and the gallbladder is opacified at thirty minutes, or as late as 1½ to 2 hours. The authors take films every five minutes for thirty minutes, and if these show the extrahepatic ducts, the film to demonstrate the gallbladder is taken at seventy minutes. The maximum opacification of the gallbladder may occur as late as four to six hours after injection. The normal common duct measures 1–5 mm. in width. The renal pelves may be visualized even when the liver is normal.

Non-visualization of the gallbladder usually indicates a surgical condition, frequently a stone in the cystic duct, or possibly an obstruction of the common duct. Stones may be visualized in the gallbladder, hepatic or common ducts.

Intravenous cholangiography is indicated in the study of the post-cholecystectomy syndrome. Stones may be demonstrated in the hepatic or common ducts. Large cystic duct stump may be demonstrated.

In cholecystitis and cholangitis the gallbladder may appear normal, may be visualized slowly, or may not be visualized. In biliary dyskinesia the commom duct may not be visualized because the opaque substance is evacuated prematurely into the duodenum. If the sphincter of Oddi is hypertonic the opacification of the common duct is prolonged.

Opacification of the extrahepatic ducts is obtained in 89 per cent of the cases. Non-visualization is noted in hepatic insufficiency, in carcinoma of the head of the pancreas with compression of the common duct and in insufficiency of the sphincter of Oddi.

Intranvenous cholangiography is indicated when the gallbladder is not visualized following oral cholecystography, when it is desirable to visualize the common duct before surgery, and in the post-cholecystectomy syndrome. The procedure is contraindicated in hepatic dysfunction, hyperthyroidism, tetany and when hypersensitivity to iodine exists.—*Charles M. Nice, Jr., M.D.*

HUGHES, E. S. R., and KERNUTT, R. H. Operative cholangiography. *Brit. M. J.*, Sept. 11, 1954, *2*, 620–623.

The authors discuss the value of operative cholangiography as noted in a review of 50 cases undergoing gallbladder surgery. They divide cholangiography into diagnostic operative cholangiography in which the injection into the common duct is made before the dissection of the biliary tree is begun, and control operative cholangiography in which the injection of contrast material is made after the common bile duct has been explored. This review was made to study the practicability of operative cholangiography as a routine procedure (1) for diagnosis of stones in the common duct; (2) to evaluate the advantage of control cholangiography over postoperative cholangiography; (3) to determine the place of operative cholangiography, in the recognition of disorders of the biliary tract co-existent with gallstones.

In the technique of the procedure it is emphasized that a uretheral catheter has definite advantages over a needle. The authors list these as: the ability of the catheter to thread the valves of Heister, measurement of the pentration by the centimeter markings on the catheter, the extra length of the catheter allowing the surgeon's hands and instruments to be removed from the field of radiation exposure. The authors use a 50 per cent solution of diodone as the opaque medium.

In discussing the place of operative cholangiography in the diagnosis of stones in the common bile duct the authors note that if the common duct were explored only in those cases in which there was clinical and surgical indication for such, 10 per cent of the stones would be missed. This is borne out by a survey of the literature. In this series cholangiography demonstrated stones in one of fourteen cases although the patient had neither clinical nor operative evidence of calculi.

The authors believe definitely that control operative cholangiography is of considerably more value that the postoperative cholangiography. Here also one case in ten demonstrated overlooked stones. These calculi are readily removed at this time without the problem of a second operation. In the opinion of the authors stenosis of the sphincter of Oddi is a very unusual occurrence. One case was classified as hypertonic sphincter as no cause for obstruction could be demonstrated. They believe that functional disorders of the biliary tract rarely require surgical treatment.

In 32 instances of these 50 cases cholangiograms appeared normal. In 12 cases the stones were visualized as filling defects and removed. In three cases no opaque medium entered the duodenum and in two of these stones were removed from the common duct. In the third case no stones could be found to explain the failure of filling of the common duct.

The authors state that diagnostic operative cholangiography is disappointing as a routine procedure but in cases of obstructive jaundice and where the common duct requires a secondary exploration, it is recommended as an important step.—*Thomas D. Allison, M.D.*

HOERR, STANLEY O. Operative cholangiography as an aid in surgery for jaundice. *A.M.A. Arch. Surg.*, Sept., 1954, *69*, 432–443.

The author discusses the advantages of cholangiography in jaundiced patients with excellent illustrative cases under the separate causative factors involved.

In obtaining these operative cholangiograms a contrast medium (urokon) is injected into the biliary tree. The method may be: (1) direct injection into the gallbladder, primarily used when the removal of the gallbladder is not anticipated; (2) injection into the common bile duct via the cystic duct, the usual method when the gallbladder has been removed; (3) direct injection into the common duct through a fine needle when the gallbladder is absent or the common duct is markedly dilated; (4) injection of the contrast material through a catheter into the common duct when the catheter is to be used as a drain at the conclusion of the operation. The author emphasizes care in technique for avoiding the usual pitfalls, *i.e.*, air bubbles, respiratory motion, superimposition of the dense contrast material over small calculi.

The causes of jaundice may include: (1) hepatic lesions (2) benign strictures of the common duct; (3) hepatic constriction of the sphincter of Oddi; (4) obstruction by malignant disease; (5) common duct calculi; (6) miscellaneous conditions. Under each of these headings the author describes interesting cases in which the cholangiograms were helpful allowing better evaluation and preventing in some cases laborious dissection in acutely ill patients.—*Thomas D. Allison, M.D.*

SAMUEL, ERIC, The radiology of vascular abdominal catastrophies. *J. Fac. Radiologists*, July, 1954, *6*, 27–37.

Retroperitoneal vascular lesions accounting for

abdominal accidents are divided according to the vessel involved and the type of pathological change as follows:

(1) Abdominal aorta: aneurysm (dissecting aneurysm and leaking aneurysm), aortic embolus and aortic thrombosis (Leriche syndrome);
(2) Splenic vessel: splenic vein thrombosis, ruptured spleen;
(3) Renal vessels: renal infarction following thrombosis or embolus.

Interperitoneal accidents involving the superior and inferior mesenteric vessels lying between layers of peritoneum include: Arterial mesenteric embolus, venous mesenteric thrombosis and intestinal strangulation.

Abdominal aortic aneurysms may give clinical symptoms either before rupture or when leaking has commenced. Rupture may occur into the peritoneal cavity or into retroperitoneal tissues. A slow leak into the retroperitoneal tissues may terminate fatally when rupture occurs into the peritoneal cavity. The recognition of this latent period while leaking is occurring is important, as aortic graft operations in the future may be life-saving in these circumstances.

Clinically these aneurysms may be asymptomatic or may present with pain radiating to either the hypochondrium, the lumbar region or buttocks, depending on the site of the aneurysm. Roentgenologically one may see a soft tissue mass of homogeneous density, lying in the line of the aorta. These aneurysms are especially prone to calcification which may extend into the soft tissue mass caused by the aneurysmal sac. Erosion of the veretebral column is relatively rare. The esophagus is apt to be displaced forward and to the left side at the diaphragmatic hiatus, this being almost a pathognomonic sign of an abdominal aneurysm near the diaphragm. A dissecting aneurysm or a slow leak from an arteriosclerotic aneurysm may present similar clinical features. Pain is the predominant symptom in both types; it is usually constant and severe. The differentiating roentgenological features of leaking from a simple abdominal aneurysm are:

(1) A soft tissue mass laying retroperitoneally and a rapid increase in the size of the abdominal mass may indicate a leak.

(2) Obliteration of the normal markings of the posterior abdominal wall and a generalized increase in the density of the retroperitoneal soft tissues.

(3) Displacement of the retroperitoneal structures due to the spread of hemorrhage into the retroperitoneal tissues sometimes causing displacement of the kidney, almost invariably in the lateral direction and best demonstrated by intravenous urography.

(4) Scoliosis of the lumbar spine with concavity toward the side of the greatest extravasation of blood, this being a relatively frequent finding.

(5) Reflex ileus, the resulting gas distention of the intestine frequently obscuring the other roentgenological signs, making the diagnosis extremely difficult.

In an *aortic embolus* lodgment of the embolus at the bifurcation of the aorta (saddle embolus) results in a dramatic clinical picture of shock, associated with obliteration of the circulation in both lower limbs. The clinical picture in such cases is usually sufficiently characteristic to make the diagnosis relatively easy and roentgenological assistance is seldom required.

In *aortic thrombosis* the symptoms are produced by a relatively slowly developing thrombosis at the bifurcation of the aorta. The presenting symptoms are usually fatigue and weariness of the lower limbs. Clinical examination will reveal the absence of pulsations in the lower limbs with palpable aortic pulsations above the umbilicus. Translumbar aortography is invaluable in the diagnosis of this condition, this demonstrating the actual site of the obstruction and the collateral circulation and enabling the condition to be distinguished from occlusive peripheral vascular disease.

Splenic vein thrombosis usually occurs as part of portal hypertension, and thrombosis of this vein is usually not readily differentiated from the underlying pathology. In other cases thrombophlebitis of the splenic vein may occur as an incident in a systemic disease, such as thrombophlebitis migrans or polyarteritis nodosa. Roentgenologically one may see enlargement and increase in density in the spleen, aneurysmal formation of the splenic artery, varicosity of the veins at the lower end of the esophagus or along the greater curvature of the stomach on barium study, or an obstruction of the splenic vein by portal venography.

Rupture of the spleen is usally a dramatic episode associated with a well established trauma, but the trauma may be relatively slight. Rupture of the spleen in such cases occurs as a delayed phenomenon. The roentgenological features of a ruptured spleen are: (a) progressive increase in the density and in the size of the spleen; (b) displacement of the gas-filled stomach and of the splenic flexure of the colon; (c) elevation of the dome of the left diaphragm; (d) serrations of the margins of the greater curvature of the stomach.

Renal infarction is usually manifested by the passage of blood in the urine and is usually unassociated with pain. Reflex suppression of renal function may occur following trauma. The absence of excretion alone is not sufficient to make a diagnosis of renal infarction. Absence of excretion of the opaque medium in the presence of a normal kidney outline, where no mechanical obstruction can be seen, suggests the possibility of renal infarction.

In *acute arterial mesenteric embolism*, an interperitoneal accident, roentgenology is called to aid in the differentiation of the condition from other abdominal catastrophies such as an acute pancreatitis, perforation of a peptic ulcer, acute intestinal ob-

struction, coronary thrombosis or a spontaneous pneumothroax. Clinically, there is acute abdominal pain, shock, rapid pulse rate, and a falling blood pressure. Abdominal distention soon appears and there is an absence of intestinal sounds from the abdomen. Roentgenologically the appearances are the result of the interference with the function and mobility of the bowel and in consequence fluid and gas collect in the dilated intestinal loops. There is increase in the motor activity of the small intestine and distention of the small intestine with the appearance of fluid levels, coming on three to five hours after the embolism. The amount of fluid present is disproportionate to the gas collected in the bowel, with the result that fluid levels tend to be relatively small and numerous. On barium studies the mucosa of the small intestine shows prominent and well-marked valvulae conniventes. Below the gas-filled loops there may be noted a soft tissue mass corresponding to the engorged and congested small intestine.

Venous mesenteric thrombosis, also an interperitoneal accident, presents none of the dramatic picture of the acute arterial embolus. The subject is a middle-aged patient who frequently gives the history of previous vascular accident, such as coronary and cerebral thrombosis, who develops progressive abdominal pain which is intermittent in character and does not radiate. Plain roentgenograms may show slight distention of the small intestine with a slight increase in the accumulation of gas and fluid present in the affected portion of the bowel. On barium study usually the affected segment of the small intestine is distended and sausage-like, and some barium may remain within this coil for a considerable period of time. This altered motor activity of a single coil of small intestine is highly significant. It is important to emphasize that the demonstration of a single coil or portion of the small intestine showing slight distention, loss of motor activity, and coarsening of the mucosal pattern is the key to the roentgenological diagnosis of venous thrombosis with infarction of the small intestine.—*Samuel G. Henderson, M.D.*

Williams, R. J. Vesico-intestinal fistula and Crohn's disease. *Brit. J. Surg.*, Sept., 1954, *42*, 179–187.

The reported case is 1 of 7 cases of vesico-intestinal fistula encountered by the author. A total of 148 reported cases of this type of fistula has been reviewed with a view of determining the etiological factors. Neoplastic fistulae account for 44 per cent of the total cases; inflammatory fistulae for 32 per cent; traumatic fistulae for 14.6 per cent and congenital fistulae for 4 per cent. Regional ileitis is an uncommon cause of vesicovaginal fistula since the author has gathered together only 16 cases.

The present case was diagnosed by means of the injection cystogram. Sodium iodide injected through the catheter failed to outline the bladder but revealed a cavity above the bladder communicating with the rectosigmoid. This cavity proved to be constituted by the terminal ileum. Barium follow-through or enema may show the causative intestinal lesion but usually not the fistula, which is generally small and not filled. The importance of resection of the involved segments of intestines in cases due to regional ileitis is stressed.

Considering vesico-intestinal fistulae in general the importance of neoplasm is emphasized; it is twice as common an etiological factor in the production of the fistulae between the bladder and distal large intestine as is diverticulitis.—*C. N. Borman, M.D.*

Erdmann, H. Indikation zur Röntgenuntersuchung des Darmes bei Lungentuberkulösen. (Indication for roentgen examination of the intestinal tract in pulmonary tuberculosis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Oct., 1954, *81*, 468–474.

Roentgenologic (Gunnar Berg) and autoptic (Källquist, Neiberding) investigations have revealed that the percentage of intestinal complications in pulmonary tuberculosis is on the decrease.

The recent European figures (12.5 per cent) are considered too low to justify routine barium examination of every known tuberculous patient. In the author's experience, modern chemotherapy often failed to cope with intestinal localizations, while surgical removal might be of help, hence the two-fold need for precise indications of examination of the intestinal tract in cases of tuberculosis. These indications are listed as:

1. "Leading" symptomatology (Höring), such as diarrhea, tenderness on presssure over definite abdominal areas at repeated examinations, localized hyperperistalsis with recurrent pain, meteorism of the splenic flexure, etc.

2. In the absence of the above symptoms, (a) if the history suggests episodes of specific peritonitis, (b) if intestinal tuberculosis was demonstrated or at least suspected at any previous time, and (c) in the presence of a recent pulmonary localization of the "secondary type," which fails to respond adequately to chemotherapy.—*E. R. N. Grigg, M.D.*

Podolsky, Morton L., and Jester, Albert W. The distribution of air in the intestinal tract of infants during the first twelve hours as determined by serial roentgenograms. *J. Pediat.*, Dec., 1954, *45*, 633–642.

The authors describe the distribution of gas in the intestinal tract of new born infants as determined by roentgenograms of the abdomen made in the anteroposterior and lateral positions at various intervals for ten to twelve hours. There was no correlation between the rapidity of the gas in the intestine and its

distribution with regard to the type of anesthesia, the duration of labor, sex or color.

All patients revealed evidence of gas in the stomach and small intestine in the first roentgenogram made as soon after birth as was possible. The majority of the patients revealed evidence of gas in the cecum and ascending colon in three to four hours after birth. All demonstrated evidence of gas in the descending colon in five to six hours. They suggest that gas probably progresses through the entire gastrointestinal tract in four to six hours after birth.

The study included 133 roentgenographic examinations on 35 newborn infants of whom 19 were premature in birth.—*James F. Martin, M.D.*

Gerber, Alex. Gastrointestinal distention in infants. *J. Pediat.*, Jan., 1955, *46*, 67–77.

The author describes three sources of gastrointestinal gas in infants: (a) food decomposition, (b) swallowed air and (c) diffusion from the blood stream. Food decomposition as a source of gas in the intestinal tract appears relatively unimportant for the major portion is CO_2 which is readily absorbed by the intestinal tract. Swallowed air appears to be the major source of gas in the intestinal tract and this occurs primarily during the act of deglutition. The author presents a series of roentgenographic reproductions on patients ranging in age from eighteen days to eight years demonstrating an increase in the gas in the stomach following the ingestion of milk which did not occur following sucking on an empty bottle for thirty minutes. The role that diffusion plays in the production of intestinal gas in infants remains uncertain. The autonomic nervous system appears important in gastrointestinal distention and the mechanism remains understood.

The author does not attempt to explain abnormal distention of the gastrointestinal tract but rather pays attention to some of the conditions in which gas is identified in the intestinal tract in a series of events which normally occur daily in the lives of these patients.—*James F. Martin, M.D.*

Hope, John W., and Gibbons, John F. Duodenal obstruction due to annular pancreas; with a differential diagnosis of other congenital lesions producing duodenal obstruction. *Radiology*, Oct., 1954, *63*, 473–490.

An annular pancreas is a band of secreting pancreatic tissue arising from the head of the pancreas and passing around the second portion of the duodenum.

About 85 cases of annular pancreas have been reported in the literature. The authors add 7 more cases with description of the roentgenographic findings.

The embryology of the pancreas is reviewed. Symptoms of high obstruction and pain vary with the degree of obstruction present.

A presumptive diagnosis should be made by the radiologist. There is usually a smooth annular narrowing of the second portion of the duodenum with dilatation of the duodenal cap. In obstructed infants, air is safely used as a contrast medium.

In differential diagnosis one should consider duodenal band, duodenal atresia, volvulus of the duodenum, and duodenal webs.

Treatment is surgcial. Generally a by-passing procedure such as a duodenojejunostomy is done.—*E. E. Seedorf, M.D.*

Ravitch, Mark M. Reduction of intussusception by barium enema. *Surg., Gynec. & Obst.*, Oct., 1954, *99*, 431–535.

Hydrostatic pressure reduction of intussuception by barium enema under fluoroscopic control is probably much less traumatic than the standard operative technique for manual reduction of an intussusception. The author fears that resection is not uncommonly necessary because the operative reduction is in itself traumatic.

Those who object to hydrostatic reduction say that gangrenous bowel may be reduced and that bowel may perforate under pressure during the reduction. In order to answer these questionable objections, experiments were done on dogs. Intussusceptions were produced in the dogs at open laparotomy and at intervals of 18, 28, 38, and 48 hours; the dogs were anesthetized and the abdomens were opened under sterile conditions. Attempts were then made to reduce the intussusception by enema under 3 feet of pressure. The author states that in no instance was gangrenous bowel reduced. There was no instance of perforation of the bowel.

It has now become the author's policy to attempt hydrostatic pressure reduction in all children regardless of the duration of the intussusception. The procedure followed in reducing an intussusception by barium enema is described in detail.

Experience has led the author to believe that the method of reduction can become more perfect with increasing experience. He notes that 90 per cent of their last 21 cases were reduced without operation. The possibility of error in diagnosis is considered and it is felt that a negative barium enema may lead to a more rapid laparotomy than might be the case without the barium enema examination.—*R. E. Kinzer, M.D*

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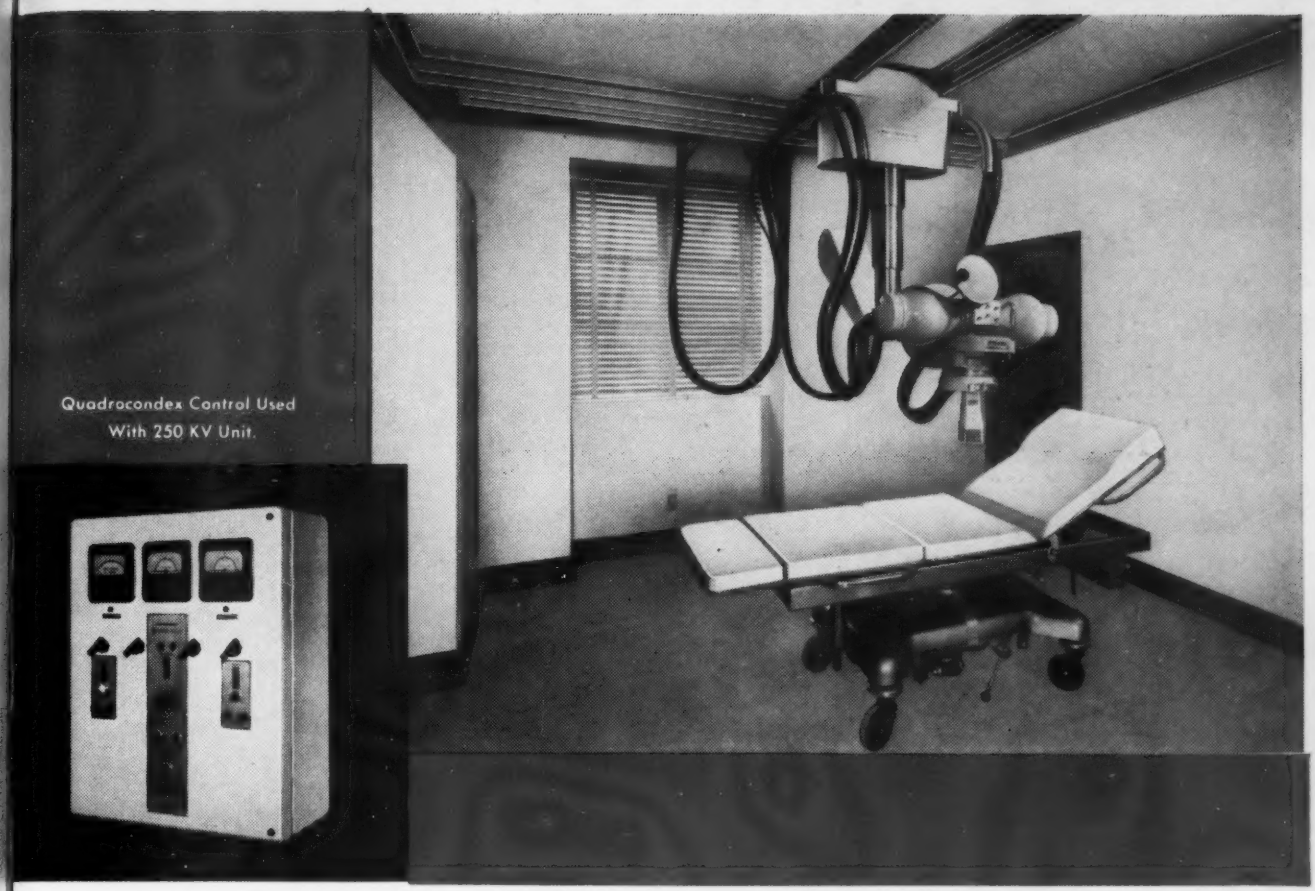

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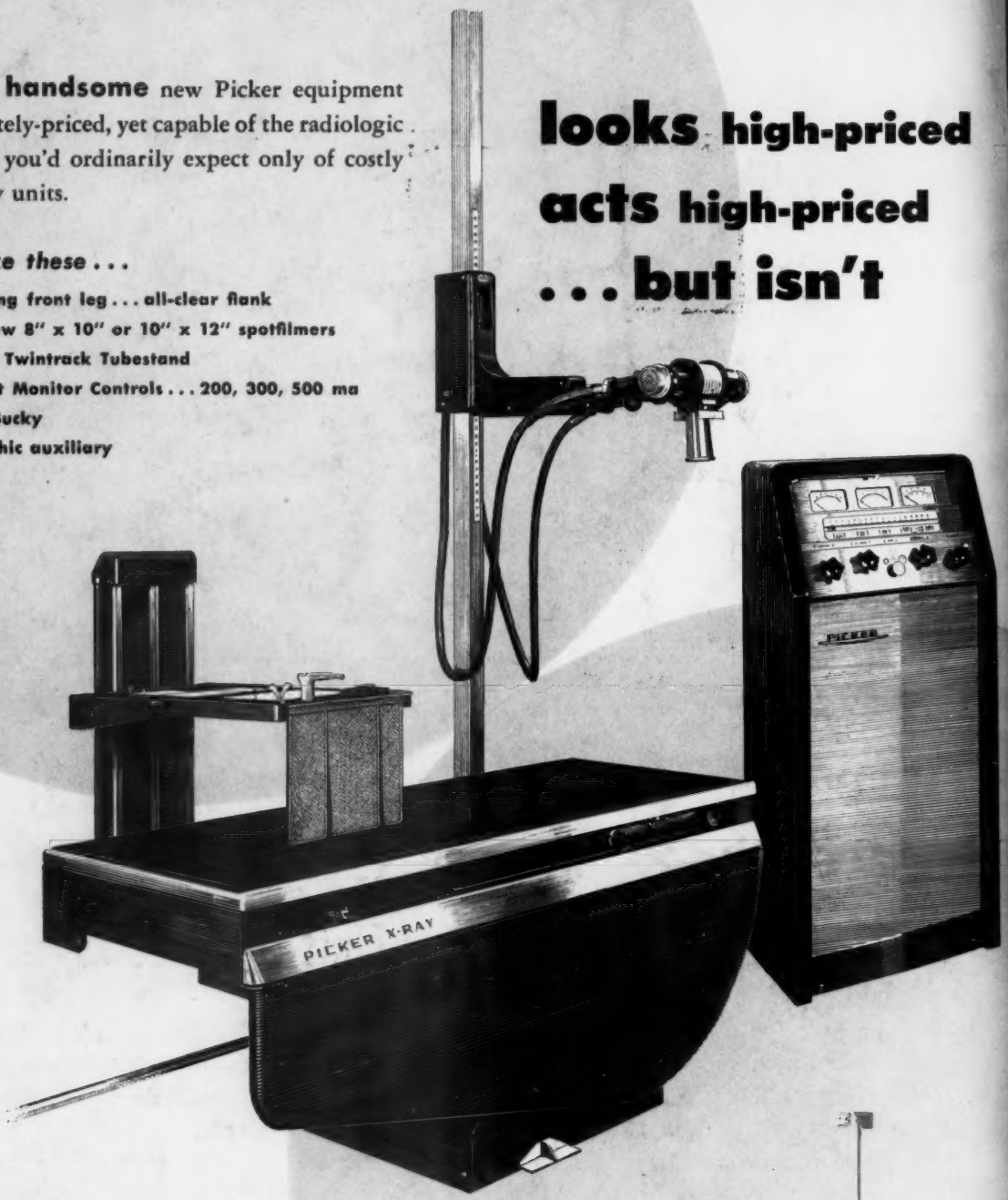
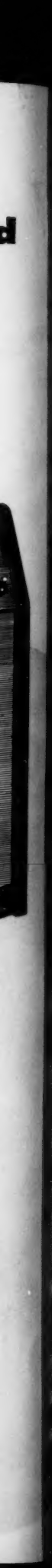
... but isn't



perfect as the "other" unit in the busy radiologist's office ... as starting equipment in the newly-opened office ... as an all-purpose x-ray resource in the small hospital or clinic. Or in any other situation where high-grade x-ray facilities are demanded, but continuous heavy-duty is not required.

new Centurion II
diagnostic x-ray unit

Picker x-ray

get the story from your Picker representative. You'll find him under "Picker X-Ray" in the classified section of your local 'phone book: or write us at 25 So. Broadway, White Plains, N. Y.